South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 22 November 1958 Weekly 2s. 6d. | Volume 32 No. 47 Deel 32 | Kaapstad, 22 November 1958 Weekliks 2s. 6d.

LUCERNE AS A CAUSE OF RESPIRATORY ALLERGY IN SOUTH AFRICA

DAVID ORDMAN, B.A., M.B., CH.B. (CAPE TOWN), D.P.H. (RAND)
The South African Institute for Medical Research, Johannesburg

An account[1] has already been given of the occurrence in South Africa of allergic vasomotor rhinitis and bronchial asthma caused by the inhalation of the dust of cereal grains. In farming practice, however, lucerne is frequently grown and is also generally handled together with cereals on the lands and in cattle feeding. As lucerne (*Medicago sativa*) is a legume and not a true cereal of the grass (*Graminae*) type like maize, wheat, oats and rye, its role as an allergen in respiratory allergy is dealt with separately in this paper.

Lucerne, a perennial legume, is one of the principal crops in the Union of South Africa and is grown almost exclusively for hay in cattle feeding. It is a good source of protein and mineral matter and is used as a feed for all classes of livestock, for which purpose the hay is often ground to meal. It is produced principally on the irrigated settlements situated on the lower Orange River, from about Buchuberg Dam to beyond Kakamas, and from those of the Vaalharts, Oudtshoorn and Graaff-Reinet areas and also on the Fish, Sundays, Modder, Sak and other rivers.[2]

Lucerne usually causes vasomotor rhinitis and bronchial asthma especially when it is handled in the dry state during baling of the hay or when milled into powder. In the farming districts of the Orange Free State, where lucerne respiratory allergy is not uncommon, it has been observed that children, mainly boys 5-10 years of age, develop mild urticaria after helping in the lucerne lands.

In the 7-year period to 1957, 57 extracts were issued as 'treatment sets' at the request of physicians for the desensitization of patients clinically sensitive to lucerne. Of these, 36 sets (63·1%) were made of lucerne extract alone and 21 sets (36·9%) contained in addition extracts of one or more cereal to which the patients were also sensitive.

In 1952 the realization that allergic sensitivity to the inhalation of lucerne dust was of significance in South Africa stimulated a close study of each case that subsequently came to notice. Information about the patients was obtained largely by correspondence with the physicians concerned. In this way from 1952 to 1957 reports were received of 38 patients with a clinical sensitivity to the inhalation of lucerne dust (confirmed by skin testing in 18 cases where this was done). In 23 patients the sensitivity was to lucerne alone and in the other 15 there was a coexisting sensitivity to one or more cereal.

Among 24 lucerne-sensitive patients whose occupation could be ascertained there were 16 farmers, 3 agricultural students working on farms, 3 workers in grain stores or mills and 2 school children.

Lucerne extracts were issued to physicians for desensitizing purposes in 30 treatment sets, either alone (19), together with a single cereal extract (6), or together with two or more cereal extracts (5).

CASE REPORTS

It was possible, from information received, to study the clinical and other details of 24 patients in whom lucerne sensitivity was a specific factor in their respiratory allergy condition. In only 6 of these, however, where desensitization with lucerne extract was actually carried out, were sufficient details of the treatment obtained for an assessment of the value of such desensitization. These cases are described hereunder, the physicians' comments being shown in italics.

Case 1

A 13-year-old schoolgirl suffered from vasomotor rhinitis and asthma from the age of 3. Her father is subject to hay fever. Her symptoms occurred only when her father—a lucerne farmer—returned home from work. When the family moved from the farm there was no further asthma until an attack occurred, when the cause was traced to lucerne being used by neighbours for feeding goats. Skin sensitivity was shown to compositae, grass pollen, house dust and lucerne. She was sensitized with the combined extracts of these substances. '*The patient can sleep with a bale of lucerne now, if she wanted to, through the night—without any adverse effect.*'

Case 2

A 16-year-old boy at an agricultural school suffered from vasomotor rhinitis with occasional attacks of asthma. His father's brother is an asthma sufferer. The symptoms were present for 3½ years and occurred only when in contact with lucerne, especially dry lucerne. Before then contact with lucerne produced watering of the eyes and some rash. Sensitivity to lucerne was confirmed by skin tests. Desensitization was carried out with lucerne extracts and the doctor reported: '*When I was about two-thirds through the course of the injections, the patient was entirely free from both hay fever and asthma despite close contact with wet and dry lucerne.*'

Case 3

A 39-year-old man managing a farm store suffered from vasomotor rhinitis. His two sons suffer from hay fever. Symptoms occurred during his daily handling of green and dry lucerne. Skin tests were not done. Desensitization was carried out with lucerne extracts. '*The patient did not complete the course of the*

injections since he was transferred to another district. He had nevertheless become completely free of symptoms.'

Case 4

A school girl aged 14 suffered from vasomotor rhinitis and asthma. She lives about half a mile from a lucerne mill and developed severe symptoms whenever the wind blew in her direction. Skin tests confirmed lucerne sensitivity. Desensitization was carried out with lucerne extracts. '*After* 10-12 *injections the patient was completely desensitized. Previously she would not have dared to go anywhere near the lands. Now she can play about in a field of lucerne without any effect.*'

Case 5

A 18-year-old farmer suffered from vasomotor rhinitis. Symptoms occurred when he worked with lucerne. Confirmatory skin testing was not done. '*Successful desensitization occurred after* 10-12 *injections of lucerne extracts.*'

Case 6

A farmer aged 29 suffered from attacks of vasomotor rhinitis and asthma when he fed sheep with lucerne. Sensitivity to lucerne was confirmed by skin tests. Desensitization was carried out with lucerne extracts. '*Patient has benefited much from the injections. Although he tries to avoid contact with lucerne as much as possible, he definitely does not get the same attacks he used to get on coming into contact with lucerne.*'

A study of the above cases shows that desensitization with lucerne extract is a highly satisfactory procedure.

Very occasionally cases occur of respiratory allergy due to the inhalation of another legume—peas (*Pisum sativum*). The pea plants which grow on farms are uprooted when the peas are mature and submitted to a crushing and sifting process, during which time respiratory symptoms have been produced by the dust.

Buckwheat (*Fagopyrum vulgare*) is neither a cereal nor a legume, but belongs to the family *Polygonaceae*, of which rhubarb is also a member. Buckwheat was introduced into South Africa for a period as a flour substitute in the local baking industry when wheat was in short supply and Ordman[3] showed that vasomotor rhinitis and bronchial asthma resulted from the inhalation of buckwheat flour in sensitive persons.

SUMMARY AND CONCLUSIONS

An investigation is reported of the occurrence of allergic vasomotor rhinitis and bronchial asthma resulting from the inhalation of lucerne dust in the Union of South Africa.

Lucerne-dust sensitivity was found mainly in farmers, but it occurred also in persons otherwise handling green or dry lucerne in stores and mills.

Details are given of 6 cases of lucerne-sensitive patients with symptoms of respiratory allergy where desensitization with lucerne extract was carried out and where the results of desensitization could be assessed.

Specific lucerne desensitization in respiratory allergy due to the inhalation of lucerne dust is shown to be very satisfactory.

REFERENCES

1. Ordman, D. (1958): S. Afr. Med. J., **32**, 784.
2. Union Department of Interior (1955): *Official Year Book of the Union of South Africa*, No. 28. Pretoria: Government Printer.
3. Ordman, D. 1947): S. Afr. Med. J., **21**, 737.

BOOKS RECEIVED : BOEKE ONTVANG

Intracardiac Phenomena. In Right and Left Heart Catheterization. A 2nd Edition, Revised and Enlarged, of *Cardiac Pressures and Pulses.* By Aldo A. Luisada, M.D. and Chi. Kong Liu, M.D. Pp. ix+179. 82 Figures. $9·50. New York and London: Grune & Stratton, Inc. 1958.

The Borderland of Embryology and Pathology. By R. A. Willis, D.Sc., M.D., F.R.C.P. Pp. ix+627+(33). 244 Figures. 101s. 3d. + 2s. 3d. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1958.

Systemic Ophthalmology. 2nd Edition. Edited by Arnold Sorsby. Pp. xiv+682+(19). 277 Figures. 118s. 3d.+2s. 3d. Postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1958.

Modern Trends in Paediatrics. Second Series. Edited by A. Holzel, M.D., D.C.H. and J. P. M. Tizard, M.A., B.M., M.R.C.P., D.C.H. Pp. xxiii+372+(13). 46 Figures. 78s. 9d. Postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1958.

British National Formulary 1957. *First Amendment* 1958. Pp. 11. 8d. net. London: The Pharmaceutical Press. 1958.

The Optimum Conditions for Viewing by Projection. By H. Hartridge, M.A., M.D., Sc.D., M.R.C.P., F.R.S. Pp. 18. 2s. 6d. Post Free. London: The Royal Photographic Society. 1958.

The First Ten Years of the World Health Organization. Pp. x+538. Illustrations. 25s. Available also in French and Spanish editions. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore, (Pty.) Ltd., P.O. Box 724, Pretoria.

Publications of the World Health Organization 1947-57. *A Bibliography.* Pp. 128. 17s. 6d. Also published in French. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore, (Pty.) Ltd., P.O. Box 724, Pretoria.

Procedures for the Testing of Intentional Food Additives to Establish their Safety for Use. Second Report of the Joint FAO/WHO Expert Committee on Food Additives. Technical Report Series No. 144. Pp. 19. 1s. 9d. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore, (Pty.) Ltd., P.O. Box 724, Pretoria.

The Agreement of Brussels, 1924, *Respecting Facilities to be Given to Merchant Seamen for the Treatment of Venereal Diseases.* Report of a Study Group. Technical Report Series No. 150. Pp. 64. 3s. 6d. Also published in French and Spanish. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore, (Pty.) Ltd., P.O. Box 724, Pretoria.

Modern Trends in Anaesthesia. Edited by Frankis T. Evans, M.B., B.S., F.F.A.R.C.S., D.A. and T. Cecil Gray, M.D., F.F.A.R.C.S. Pp. ix+318+(13). 30 Figures. 76s.+ 1s. 9d. Postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1958.

John Wesley among the Physicians—A Study of Eighteenth-Century Medicine. By A. Wesley Hill, B.A., M.B., B.Ch. Pp. 135. 10s. 6d. net. London: The Epworth Press. 1958.

Ciba Foundation Colloquia on Ageing. Volume 4. *Water and Electrolyte Metabolism in Relation to Age and Sex.* Editors for the Ciba Foundation. G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch. and Maeve O'Connor, B.A. Pp. xii+327. 85 Illustrations. 45s. net. London: J. & A. Churchill Ltd. 1958.

Developmental Potential of Preschool Children. An Evaluation of Intellectual, Sensory and Emotional Functioning. By Else Haeussermann. Pp. xvii+285. 19 Figures. $8·75. New York and London: Grune & Stratton, Inc. 1958.

A Primer in Medical Technology. By Paul M. Kraemer. Pp. viii+338. 33 Figures. 58s. 6d. Oxford: Blackwell Scientific Publications. 1958.

Advances in Clinical Chemistry. Edited by Harry Sobotka and C. P. Stewart. *Volume I.* Pp. xi+398. 9 Figures. $12·00. New York and London: Academic Press Inc. 1958.

Metabolic Disturbances in Clinical Medicine. Edited by G. A. Smart, B.Sc., M.D., F.R.C.P. Pp. viii+358. Illustrations. 45s. net. London: J. & A. Churchill Ltd. 1958.

South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL : VAN DIE REDAKSIE

SUBARACHNOID HAEMORRHAGE

In a thoughtful Lumleian lecture Dr. Elkington[1] has included spontaneous subarachnoid haemorrhage in his review. Conservative treatment of this malady carries a high mortality of 45-60%. After the age of 40 the mortality increases with age. In an attempt to reduce this mortality Elkington advocates that bilateral carotid angiography should be carried out as soon as possible to demonstrate the cause of the haemorrhage and allow surgical treatment. There is by no means complete agreement on the question of exactly when this should be done. McKissock and Walsh[2] hold that it should be performed immediately, provided each common carotid artery in turn can be compressed without ill effect. Of over 400 cases in which angiography was performed during the acute phase[3,4] arterial (berry) aneurysms were shown to be the cause of haemorrhage in 54%, arteriovenous malformations in 11·5%, primary intracerebral or intracerebellar haemorrhage in 7·5%, while no lesions was demonstrated in 27%. It should be noted that post-mortem evidence in any large series indicates that cases with berry aneurysms constitute about 75% of the total. These authors emphasize the need for demonstrating the precise cause of the bleeding so that it can be dealt with surgically, either directly at the site of the defect itself or by some form of carotid ligation.

The anatomical site is also of importance in prognosis, as was shown by Logue[5] in aneurysms of the anterior cerebral and anterior communicating arteries. In these arteries the first bleed seldom kills, but there is a tendency to bleed again with fatal outcome, especially in the second week. The time factor is the factor of major importance.

McKissock and Walsh[2] operated on 52 patients who were in danger of dying from the haemorrhage which caused their admission, and these included all cases in coma and semicoma and those with severe neurological signs. Their mortality in this group was 65·5% whereas in 43 cases of the same grade of severity, treated conservatively, the mortality was 90%. Those who were admitted within 8 weeks of the haemorrhage and were recovering from it, and were therefore in danger of recurrent haemorrhage, formed their second group. In 72 such cases treated surgically the mortality was 12·5% while it was 37·5% in 48 cases treated conservatively. If a period of more than 8 weeks has elapsed since the initial bleed[6] there is only a 20% risk of recurrent haemorrhage, and in 17 cases treated surgically the mortality was 23·5%, while with conservative treatment it was 6%.

These figures all apply to demonstrated arterial aneurysms and it seems that if more that 8 weeks have elapsed since the haemorrhage, conservative treatment is preferable, but that surgery should be undertaken if the period is less than this, provided an aneurysm is demonstrated in a suitable site. Not all would agree to operate during the phase of acute haemorrhage; Elkington points out that with the use of artificial hypothermia and improved surgical techniques early operation will become more frequent. Using artificial hypothermia Botterell *et al.*[7] report a mortality of only 30% in 64 patients operated on within 7 days of subarachnoid haemorrhage.

Prognosis in the angiomatous group is good and surgical treatment is likely to result in cure since the tendency to bleed again is small. It also seems likely that the prognosis is better in the group in which no lesion is demonstrated by angiography.

1. Elkington, J. St. C. (1958): Lancet, **2**, 327.
2. McKissock, W. and Walsh, L. (1956): Brit. Med. J., **2**, 559.
3. McKissock, W. (1956): Ann. Roy. Coll. Surg. Engl., **19**, 361.
4. Walsh, L. S. (1956): Acta radiol. (Stockh.), **46**, 321.
5. Logue, V. (1956): Brit. Med. J., **1**, 473.
6. Hyland, H. H. (1950): Arch. Neurol. Psychiat., **63**, 61.
7. Botterell, E. H. (1958): J. Neurosurg., **15**, 4.

BLOEDBESINKING

Die ondersoek van bloedbesinking is seker een van die eenvoudigste spesiale ondersoeke in algemene gebruik, en ewe akkuraat uit te voer in die navorsingsinrigting en die verafgeleë spreekkamer in die platteland. Die geskiedenis van die toets is interessant; sy diagnostiese waarde is gering maar sy praktiese waarde is groot.

Biernaki (1891) was die eerste om die verskynsel in siektetoestande te bestudeer.[1] Fähreus het in 1918 sy waarnemings oor die besinking van eritrosiete gepubliseer, soos gevind in swangerskap,[2] en daarna het hy sy belangstelling uitgebrei na mediese geskiedkunde, en die aandag gevestig op die *crusta inflammatoria* of *phlogistica* van die ou Griekse geneeshere. Die 4 ‚humors' van die oergeneeskunde is in die besinkingsbuis te besigtig, nl: Die flegma, of witlagie, wat as die oorsaak van die siekteproses beskou is, die bloed, en heel onder die ‚swart gal'—die massa gepakte rooibloedselle. Die serum kom ooreen met die ‚geel gal'. Die verhouding van die 4 humors het die basis van die patologie en die patogenese van die outyd gevorm, en verklaar die populariteit van bloedlating oor 'n tydperk van ruim 2,000 jaar.[3]

Die suspensie-stabiliteit van die bloed is afhanklik van die vermoë tot *rouleaux* formasie van die rooibloedselle, 'n meganisme wat nog nie ten volle verklaar is nie. Waarskynlik speel fibrinogeen 'n belangrike rol en om dié rede beïnvloed die fibrinogeen-gehalte die besinking merkwaardig. Die proteïen-fraksies in die bloed is ewe belangrik, indien nie meer belangrik nie. Fibrinogeen, globulien en albumien

het in hierdie volgorde effek op die hoogte van die lesing—albumien het in werklikheid 'n negatiewe korrelasie.[2] 'n Formule bestaan waarvolgens die besinking vanaf genoemde 3 proteïenwaardes, bereken kan word met 'n akkuraatheid van binne 20% deviasie. Die grootte van die rooiselle, die pH en oksigenasie van die bloed, asook die aantal rooiselle, beïnvloed die besinking. Die temperatuur en die lesitien-cholesterol verhouding is ook belangrik.[1,3]

Die besinking in anemie is wisselvallig en moet gesien word as die totaal van verskeie faktore: (1) Soos die rooiselle verminder, so styg die besinking—'n basiese fisiese eienskap. (2) Faktore bestaan in anemie wat die effek volkome kan neutraliseer, bv. in 'n mikrositiese anemie sal die kleinheid van die selle hulle stadiger laat sink. Verdunningseffekte verdun insgelyks die plasmaproteïene en vertraag die besinking. Gewoonlik egter is die besinking effe hoër in anemie, maar hierdie faktore kan tot 'n onverwagte hoë òf lae besinking in gevalle met anemie lei. 'n Hoë besinking in 'n anemiese pasiënt is dus nie *noodwendig* die gevolg van die anemie nie.[2]

'n Ander toestand waarin die besinking ook versigtig beoordeel moet word, is in gevalle met sianose. Die verhoogde koolsuurgas-inhoud lei tot swelling van die rooiselle en vertraag die besinking. Om hierdie rede is die besinking dikwels laag in hartversaking en sianose, hoewel in die eersgenoemde geval die lewerstuwing 'n effek op die proteïene mag hê, en dus deels verantwoordelik is vir dié verskynsel.

In akute infeksies word die temperatuurstyging eerste gevind en daarna 'n styging in die witseltelling. Die besinking is selde in die eerste 24 uur verhoog. Hoewel klinies volkome genesing teenwoordig mag wees, bereik die besinking sy piek as die temperatuur eers normaal is en mag dit nog etlike weke verhoog bly as gevolg van metaboliese versteurings in die proteïen-metabolisme. In toestande met weefselnekrose styg die besinking ook, en dan gewoonlik langsamer as in akute infeksies bv. in gevalle met miokardiale infarksie. Die grootste belang van hierdie toets skuil in sy gebruik in gewrigstoestande—'n verhoging dui op aktiwiteit van die proses of op komplikasies.[2] In tuberkulose sal 'n buitengewone snel besinking op 'n komplikasie soos akute pleurale of renale aantasting dui. In aktiewe tuberkulose is die besinking matig versnel, hoewel 'n normale besinking in 2·1% van gevalle van ope tuberkulose gevind is.[2]

Hoewel hierdie toets dus nie spesifiek is nie, behalwe in een geval, het hy die toets van die tyd deurstaan as 'n nuttige prosedure by alle pasiënte. As die geneesheer dus bewus is van die beperkings en die faktore wat dit beinvloed, styg die waarde van die toets dienooreenkomstig. Die geval waarin die toets spesifiek is, is in sekelselanemie, waar 'n verskil van meer as 20 mm. in die besinking van veneuse en geoksigeneerde bloed gevind word.[3]

1. Best, C. H. en Taylor, N. B. (1955): *The Physiological Basis of Medical Practice*, 6e Uitgawe. Baltimore: Williams en Wilkins.
2. Wintrobe, M. M. (1956): *Clinical Haematology*, 4e Uitgawe. Londen: Henry Kimpton.
3. Winsor, T. en Burch, G. E. (1944): Amer. J. Med. Sci., 207, 152.

ANALGESIA IN PANCREATITIS WITH SPECIAL REFERENCE TO THE EFFECT ON PRESSURE IN THE COMMON BILE-DUCT*

G. EFRON, M.B., CH.B. (CAPE TOWN), *Surgical Registrar, Groote Schuur Hospital, Cape Town*

The agonizing pain of acute pancreatitis requires a potent analgesic for its relief and in the past morphine was recommended. That morphine causes spasm of the sphincter of Oddi has been shown, amongst others, by Prof. J. H. Louw,[1] Butsch *et al.*[2] and Nossel and Efron.[3] This fact is significant because, firstly, one of the more popular current theories of the aetiology of acute pancreatitis is that of biliary reflux up the pancreatic duct as a result of spasm of the sphincter of Oddi.[4] Secondly, it has been shown that one of the causes of pain in pancreatitis is the raised intraductal pressure due to spasm of the sphincter of Oddi.[5] Whether one favours the biliary-reflux theory or not, Nossel and Efron[3] showed that morphine may cause a rise of serum amylase in a normal person (2 cases out of 43) and that the probable mechanism of this rise was spasm of the sphincter of Oddi.

This investigation was carried out in an attempt to find a suitable analgesic agent for the relief of pain in acute pancreatitis, with special reference to possible side-effects on pressure in the common bile-duct.

Material and Methods

The investigations were carried out on patients who had undergone cholecystectomy for gall-bladder disease and in whom T-tube drains had been left in the common bile-duc after exploration. None of these patients had any history suggestive of pancreatitis and in all of them the short limb of the T-tube was at least $\frac{1}{2}$ inch away from the ampulla of Vater. All the cases were investigated approximately 10 days after the operation after patency of the common bile-duct was demonstrated by cholangiography.

Apparatus Used

The pressure apparatus (Fig. 1) consists of a water manometer which is connected (1) to the T-tube emerging from the abdominal wall and (2) by means of a Y-tube to a vacolitre of normal saline, used (*a*) to fill the system and (*b*) to increase the ductal pressure, if necessary. In this way the pressure required to overcome the sphincter tone is determined.

The manometer and tubing are filled with saline by the drip and connected to the T-tube. The drip is then clamped off. Throughout the experiment the patient lies at ease and restful in bed. Once the level in the manometer is constant (there is a variation of the meniscus with respiration of ± 1 cm.), a reading is taken. The patient is now given amyl nitrite to inhale, which completely relaxes the sphincter of Oddi. The pressure falls from 8 to 15 mm. of water and a reading is taken again. The latter reading is regarded as the zero line. After a few minutes the pressure rises again to approximately the level before the amyl nitrite was inhaled. The difference between this pressure and the zero level is called the 'resting intrabiliary pressure' which is thought to

* A paper read at the First Congress of the South African Association of Surgeons, Cape Town, April 1958.

Fig. 1. The apparatus used for measuring intrabiliary pressure.

be due to the normal tone of the sphincter of Oddi. The patient is then given the drug to be tested and the readings recorded.

Intrabiliary Pressures

The graph in Fig. 2 shows the effect of morphine on the sphincter of Oddi. The resting intrabiliary pressure was

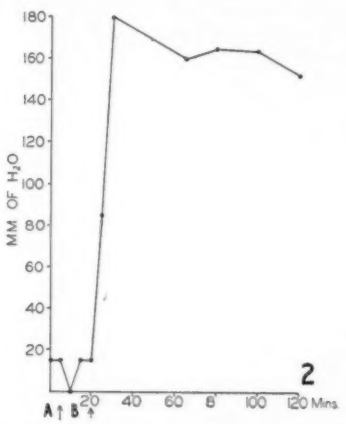


Fig. 2. Graph showing the effect of morphine on the intrabiliary pressure. A=amyl nitrate. B=morphine gr. $\frac{1}{6}$.

15 mm. of water. Morphine, 1/6 gr., was injected intramuscularly. The pressure started to rise within about 5 minutes and reached a peak of 180 mm. after 15 minutes. The rise in pressure lasted about 2 hours.

The next experiment was an attempt to show that when a rise in serum amylase is produced by morphine, it is due to spasm of the sphincter of Oddi. One of the patients, in whom it was found that morphine produced an abnormally raised serum amylase, was explored for stone in the common bile-duct and a T-tube left in her common duct. Post-operative intrabiliary pressures were measured at frequent intervals and serum-amylase levels were determined $\frac{1}{2}$ hour before and $2\frac{1}{2}$ hours and $3\frac{1}{2}$ hours after an injection of morphine.

Two days later the test was repeated with the drip opened and the pressure of the fluid in the bile duct raised to between 350 and 450 mm. of water, till the saline flowed steadily through into the duodenum at a rate of 60 drops per minute—in this way maintaining patency of the sphincter of Oddi. The serum-amylase values were again determined before and after an injection of morphine. In Table I the results are shown of the simultaneous studies on bile-duct pressures

TABLE I. THE RESULTS OF BILE-DUCT PRESSURES AND SERUM-AMYLASE VALUES

	Elevation of Bile-Duct Pressure in mm. of water	*Duration of Elevation of Pressure (minutes)*	*Serum Amylase*		
			Premed	*2½ hrs.*	*3½ hrs.*
1	140–160	180	8μ	56μ	56μ
2	345–455 (Perfusion pressure)	150	8μ	8μ	8μ
3	140–160	170	8μ	52μ	46μ

Morphine $\frac{1}{6}$ gr. given hypodermically

and serum-amylase values (with and without perfusion of the common bile-duct) after an injection of morphine. (Serum-amylase values were determined according to Wohlgemuth's method. Normal values for our laboratory are 3-10 units per c.c.)

After an injection of morphine, the rise in serum amylase was abolished by maintaining complete patency of the sphincter of Oddi. From this it appeared that a rise in the serum amylase produced by morphine was probably due to its mechanical action on the sphincter of Oddi and not to any effect on the secretory activity of the gland. It was therefore apparent that morphine, or any drug that causes spasm of the sphincter of Oddi, was unsuitable as an analgesic in the treatment of acute pancreatitis, and that the ideal drug, or combination of drugs, should be a potent analgesic as well as an antispasmodic of the sphincter of Oddi. The purpose of this work was to find such a drug. Many substances and combinations were tried, some of which will be dealt with here.

Analgesic Drugs Used

1. *The opiates* all behaved in the same fashion as morphine. Twelve experiments were done with morphine—the average rise in pressure was 110 mm. of water. Omnopon was used in one patient. The pressure started rising after 5 minutes and reached a peak of 80 mm. of water after 50 minutes.

2. *Pethidine and physeptone*, which are said to have the central analgesic effect of morphine without the spasmodic effect on the sphincter of Oddi, were also tried. Both these drugs are less potent analgesics than morphine and their effect on the sphincter of Oddi and on intrabiliary pressure is indicated in Fig. 3. Both pethidine and physeptone cause

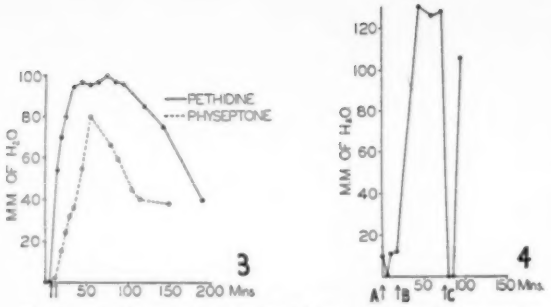


Fig. 3. Graph showing the effect of pethidine and physeptone on intrabiliary pressure.
Fig. 4. Graph showing the effect of amyl nitrite on spasm of the sphincter of Oddi produced by morphine. A=amyl nitrite. B=morphine. C=amyl nitrite.

a rise in intrabiliary pressure. With pethidine the intrabiliary pressure rose to a maximum of about 100 mm. of water in 20 minutes and started to fall after 95 minutes. Radiologists are aware that pethidine causes spasm of the sphincter of Oddi and give intravenous pethidine to produce this spasm in doing biligrafin studies. Physeptone, a poorer analgesic, produces a slower rise in intrabiliary pressure, which starts to fall after 1 hour.

Combinations of Analgesics and Antispasmodics

(i) *Morphine and amyl nitrite* (Fig. 4). Amyl nitrite produces immediate and complete relaxation of the spasm (of the sphincter of Oddi) produced by morphine. This relaxation is short-lived and the pressure rises once the inhalations are stopped. Unfortunately amyl nitrite cannot be inhaled for a long period of time because of unpleasant side-effects.

(ii) *Morphine and glycerine trinitrate.* Glycerine trinitrate is not as effective an antispasmodic as amyl nitrite and, unless a large dose is used, one does not get much relaxation of the sphincter of Oddi.

(iii) *Morphine and atropine.* This combination is recommended by many on the assumption that the atropine acts as an antispasmodic. Atropine does not appear to have any effect on the sphincter of Oddi (Fig. 5). It certainly does not overcome or prevent the marked spasm and increase in intrabiliary pressure produced by morphine. The use of atropine as a biliary antispasmodic therefore appears ill-founded.

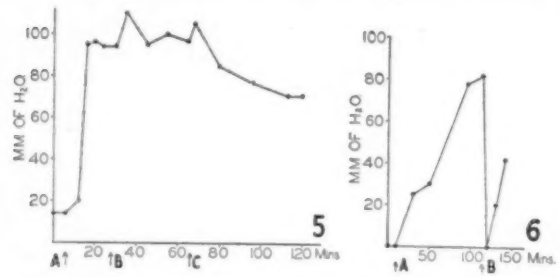


Fig. 5. Graph showing the effect of atropine and aminophyllin on intrabiliary pressure following an injection of morphine. A=morphine. B=atropine. C=aminophyllin.
Fig. 6. Graph showing effect of an injection of morphine and aminophyllin given simultaneously on intrabiliary pressure. A=morphine and aminophyllin. B=amyl nitrite.

However, there is place for atropine, or related substances such as probanthine, in the treatment of acute pancreatitis, *viz.* as vagal secretory inhibitors.

(iv) *Morphine and aminophyllin.* Intramuscular aminophyllin was used in this experiment. Aminophyllin appears to maintain partial relaxation of the sphincter after an injection of morphine for ±1 hour, after which the intrabiliary pressure rises (Fig. 6). The disadvantage of intramuscular aminophyllin is that it is a painful injection.

The other combinations of drugs tried were as follows: Morphine and khellin, morphine and papaverine, morphine and priscol, morphine and daptazole, morphine and scopolamine and morphine and ephedrine. All these antispasmodics were unable to overcome the increase in intrabiliary pressure produced by morphine. The combination of morphine and nalorphine was also tried (Fig. 7). The nalorphine was injected 40 minutes after the injection of morphine. The nalorphine relaxes the spasm produced by morphine, but unfortunately it neutralizes the analgesic effect of morphine as well.

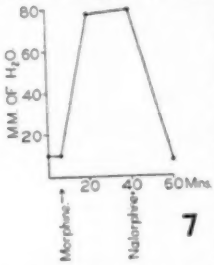


Fig. 7. Graph showing the effect of nalorphine on intrabiliary pressure following an injection of morphine.

The effects of amyl nitrite, glycerine trinitrate, aminophyllin and atropine were tried on the spasm of the sphincter of Oddi produced by pethidine and physeptone. The results were very similar to that obtained with morphine.

AVAFORTAN

At this stage a new drug, avafortan, was received for clinical trial. A few facts about avafortan, as supplied by the manufacturers, are relevant: Avafortan is free from alkaloids and consists of 2 components: avapyrazone and pyrazolone. Its chemical formula is as follows:

(1) Iso amyl ∝[N-(β diethylaminoethyl)]-amino-phyl acetate-bis-phenyldimethyl-pyrazolone-methyl-amino-methane-sulphonate (avapyrazone).
(2) Sodium-phenyl-dimethyl-pyrazolone-methylamino-methane-sulphonate (pyrazolone).

(i) The avapyrazone has an anticholinergic action—it relieves spasm of smooth muscle in the bile ducts, urinary tract, bronchi and blood vessels (coronary blood flow is markedly increased). It is interesting to note that it relieves bronchospasm, which may be a prominent feature of acute pancreatitis. We had such a case in the wards recently.

(ii) The pyrazolone is a central analgesic and it is stated that it also has an anti-inflammatory action, which may be important in the treatment of spasm due to inflammation.

Toxicity and tolerance. Avafortan is well tolerated with a wide margin of safety. Unlike the opiates and their synthetic substitutes it is stated that avafortan will not mask the acute abdomen. I have not come across any untoward effects of the drug. It can be given intramuscularly, intravenously and by mouth. I used the drug mainly intramuscularly (4-5 c.c.) and found this to be a painless injection.

Tests with Avafortan

Avafortan was given to a large number of patients who had been on various analgesics for extensive carcinoma,

Buerger's disease, etc. and it was concluded that 4 c.c. of avafortan given intramuscularly was a slightly less potent analgesic than 100 mg. of pethidine. The analgesic and antispasmodic effects were determined in numerous cases of renal colic in the hospital. The results were excellent.

The effect of avafortan on the sphincter of Oddi and on intrabiliary pressure was tested on a male patient who had undergone cholecystectomy and exploration of the common

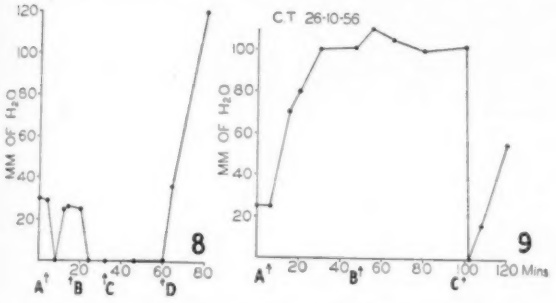


Fig. 8. Graph showing the effect of avafortan on resting intrabiliary pressure. A=amyl nitrite. B=avafortan. C=amyl nitrite. D=morphine.
Fig. 9. Graph showing the effect of avafortan on intrabiliary pressure after an injection of morphine. A=morphine. B=avafortan. C=amyl nitrite.

bile-duct (Fig. 8). The resting intrabiliary pressure was 30 mm. The patient was given amyl nitrite to inhale and the pressure fell to zero; 5 minutes later the pressure rose to 25 mm. of water. The patient was given 4 c.c. of avafortan intramuscularly and the pressure fell to zero after about 10 minutes. Five minutes later the patient was again given amyl nitrite to inhale and the pressure remained at zero. After 1 hour the patient was given 1/6 gr. of morphine intramuscularly. The pressure rose to 120 mm. of water after 20 minutes.

In another patient who was given avafortan there was no effect on the intrabiliary pressure. One hour later $\frac{1}{4}$ gr. of morphine was given and after 10 minutes the pressure started rising.

In a 3rd test (Fig. 9) the patient was given $\frac{1}{4}$ gr. of morphine intramuscularly. The pressure rose in the manometer to 100 mm. after 30 minutes. After a further $\frac{1}{4}$ hour, 4 c.c. of avafortan was given intramuscularly with no effect on the pressure. One hour after the injection of the avafortan the pressure was still at 100 mm. of water. At this stage the patient inhaled amyl nitrite. The pressure dropped to zero after 3 minutes. Ten minutes after the inhalation of amyl nitrite the pressure started rising again and went up to 50 mm. of water.

From these tests it was concluded that avafortan did not cause spasm of the sphincter of Oddi, perhaps had a slight antispasmodic effect on the sphincter, but was unable to release the spasm of the sphincter produced by morphine.

Clinical Trials with Avafortan

Clinically avafortan has been used in 19 cases of acute pancreatitis. In 16 cases the pain was relieved and in the other 3 partial relief was obtained. In no case was the pain aggravated. In 2 cases of acute cholecystitis, one patient obtained complete relief and the other had no relief. In 2 patients with biliary colic 1 case had relief after avafortan with the first attack but not subsequently. The other patient had no relief of pain. Perhaps all the patients who were unrelieved of their pain should have had intravenous avafortan as recommended.

A further observation was recently made with avafortan tablets. In a European female aged 79 a routine post-operative cholangiogram was made on the 10th day after an exploration of the common bile-duct for stone. The patient was symptomless. On screening, marked spasm of the sphincter of Oddi was found. It took a fair amount of pressure to overcome the spasm. The lipiodol flowed through into the duodenum after 3 minutes. The next day the intrabiliary pressure was measured (Fig. 10). The resting pressure was 20 mm. water. After amyl-nitrite inhalation the pressure fell to zero. Five minutes later the pressure rose to 15 mm. water. One tablet of avafortan was given and 2 hours later the pressure was zero; 4 hours later it was still zero. At this stage the cholangiogram was repeated. On this occasion much less pressure was required to inject the dye, which appeared in the duodenum after 15 seconds.

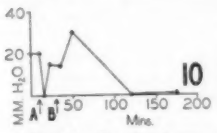


Fig. 10. Graph showing the effect of an avafortan tablet on intrabiliary pressure in a patient with spasm of the sphincter of Oddi. A=amyl nitrite. B=avafortan.

DISCUSSION

The above results raise interesting possibilities.

Firstly, if chronic relapsing pancreatitis is due to biliary reflux as a result of spasm of the sphincter of Oddi, should not the treatment of this condition be medical and sphincterotomy be replaced by a suitable antispasmodic? I have tried avafortan on one such case, who has done well. *Secondly*, avafortan may be the answer to so-called biliary dyskinesia (which is said to be due to spasm of the sphincter of Oddi).

It would appear that we have no ideal drug which simultaneously is a potent analgesic and an antispasmodic of the sphincter of Oddi for use in the treatment of acute pancreatitis. The disadvantages of the opiates and their derivatives, both alone and in combination with various antispasmodics, have been shown.

It appears that avafortan, although not the most potent analgesic available, does not produce spasm of the sphincter of Oddi and thus is the drug of choice at present.

We must search for a drug which will be an analgesic with a more powerful antispasmodic effect on the sphincter of Oddi. Such a drug will possibly replace the operation of sphincterotomy in the treatment of chronic relapsing pancreatitis and biliary dyskinesia.

SUMMARY

1. The effect of various analgesics alone and in combination with various antispasmodics on common-bile-duct pressure has been shown.

2. The results of a clinical trial of avafortan as an analgesic and antispasmodic are analysed.

3. The need for a more powerful analgesic and antispasmodic of the sphincter of Oddi is indicated, with particular reference to the treatment of pain in acute pancreatitis.

4. It is postulated that such a drug may be of value in the

treatment of cases of biliary dyskinesia and in chronic relapsing pancreatitis due to spasm of the sphincter of Oddi.

I wish to thank Prof. J. H. Louw for his encouragement, valuable advice and help, Prof. D. J. du Plessis for his interest and advice, and Messrs. Noristan Laboratories of Silverton, Pretoria, for the supplies of Avafortan.

REFERENCES

1. Louw, J. H. (1946): *The Morphology of the Main Bile Ducts*. Ch.M. Thesis, Medical Library, University of Cape Town.
2. Butsch, W. L., McGowan, J. M. and Walters, W. (1936); Surg. Gynec. Obstet., **63**, 451.
3. Nossel, H. and Efron, G. (1955); Gastroenterology, **29**, 409.
4. Doubilet, H. (1957); Proc. Roy. Soc. Med., **50**, 629.
5. Card, W. I. (1957); *Ibid.*, **50**, 637.

HYPOTHERMIA: ITS USES IN MODERN CARDIOVASCULAR SURGERY

DENIS N. FULLER, F.R.C.S., *Thoracic Surgeon, Johannesburg*

Since hypothermia is now being used as an aid in cardiovascular surgery, it is appropriate at this time to review the extensive literature on the subject and present the relevant facts and opinions to the medical profession in South Africa. As this subject has not been reviewed previously in this country, I have quoted the authorities rather extensively.

The word 'hypothermia' is of course not synonymous with 'freezing', a word sometimes used for it in the lay press, but implies a cooling of body temperature by several degrees centigrade only.

PHYSIOLOGY

Cooling delays the onset of anoxia during interruption of oxygen supply and postpones secondary enzymatic changes that follow anoxia and lead ultimately to death of the cell. If an animal is placed in a cold environment it combats the tendency to a fall in body temperature by shivering. The adrenal and pituitary glands react as they would do under any severe stimulus, by an outpouring of their metabolic hormones. If these reactions are minimised or prevented by the use of drugs or by anaesthesia, then cooling occurs at a more or less even rate and *pari passu* there is a diminution in oxygen requirement. All the body reactions are slowed down. At first there may be very little evidence of physiological change, but around 32°C consciousness is lost and the breathing and heart rates slow down. At lower temperatures the activity of the various organs may cease. Cardiac arrhythmias are common between 28° and 30°C and ventricular fibrillation, which is one of the gravest complications, may develop. The lower the temperature the greater the chances of fibrillation occurring and around 25°C it is present in a high proportion of cases because the hypothermic heart still requires oxygen in order to function. Below 25°C the coronary flow is disproportionately high in spite of falling aortic pressure. Acceleration of the heart rate during hypothermia lessens coronary flow; an increase from 50 to 70 beats a minute at 20°C completely arrests coronary flow (Brock[1]). If the coronary flow can be maintained artificially and the work load on the heart reduced to a minimum, cardiac arrest may be postponed until a temperature of 14°C is reached. Very low temperature may cause tissue damage apart from the anoxic effect. At 25°C the circulation can be occluded safely for 15 minutes without there being any clinical evidence of tissue damage; at 28°C the period is about 10 minutes.

The metabolism of glucose ceases at 31°C and during hypothermia operations this must be remembered. The intravenous drip should contain half-normal saline or blood. Sodium citrate is also not metabolized and may reach concentration high enough to be toxic to the myocardium. Likewise, stored blood with its high potassium-ion concentration may produce cardiac arrest.[2]

The optimum temperature of tissue functioning is brought down by a drop of the potassium content in the extracellular medium and an increase in the calcium. The ultimate effect of hypothermia down to 30°C is hypometabolism (40%), bradycardia (60-80 per minute), hypotension (80-100 mm. Hg), bradypnoea (12 per minute), hypokalaemia, alkalosis and moderate hypercalcaemia, hypocoagulability (50-60% prothrombin index) and moderate hyperglycaemia. The ECG shows slowing of the heart rate, progressing to prolonged electrical systole and heart block. Auricular fibrillation is common around 32°C and ventricular fibrillation at 28°C. This is particularly apt to occur during the re-warming.

Breathing stops at varying temperatures. Muller[3] noted that two infants breathed when their temperatures were 19 and 19·5°C respectively and Kaplan[4] had a child cry at 24°C. In Glen's series,[5] spontaneous respiration ceased at 28°C but sometimes at 25°C. At 25°C no animal survived complete circulatory occlusion for 20 minutes. However, he clamped off both brachiocephalic arteries in dogs for 30-55 minutes and all recovered normally. He totally exsanguinated one dog and re-introduced the blood. This too recovered. With the depression of respiration an acidosis develops. Delorme[6] thinks that hypercapnia (excess CO_2) acts as an innocuous metabolic depressant enabling warm-blooded animals to survive temperatures far below the lethal level if oxygen is breathed. Likewise Holmdahl[7] showed that dogs exhibit some high tolerance to respiratory acidosis under hypothermia. He postulated that the sudden elimination of carbon dioxide may threaten cardiac action. However, most workers prefer an alkalosis to an acidosis. The hypocoagulability which in Lewis's experience may be manifest by an increased tendency to post-operative haemorrhage[8] is thought by Huguenard[9] to be due to sympathalase.

PRODUCTION OF HYPOTHERMIA

The following methods of lowering body temperature have been used: (*a*) Drugs, (*b*) surface cooling, (*c*) vascular cooling and (*d*) internal cooling and (*e*) a combination of two or more of these.

(*a*) *Drugs*

These have been pioneered by French workers (notably Laborit) to produce hypometabolism. The so-called 'lytic cocktails' produce multifocal inhibition of the autonomic nervous system, both central and peripheral (neuroplegia). The essential ingredients of such a cocktail should possess antiadrenergic (chlorpromazine), anticholinergic (pethedine, atropine), and antihistiminic (phenergan) properties. With these it is possible to obtain results as with a 'balanced anaesthetic' comprising barbiturate, a curarizing agent and a ganglion-blocking agent (vasoplegia). Bimar[10] used hydergine and diethazine in combination instead of chlorpromazine.

Hydergine is a vasoplegic with a central (medullary) effect. It inhibits the carotid-sinus and other presso-sensory reflexes. It induces latent adrenosympatholysis by inhibiting the effects of adrenalin in the case of sympathetic stimulation.

Diethazine has a vagolytic central action and hypnotic effect and is a depressor of the motor centres. It is also cholinergic and antihistaminic. These properties make its respiratory and circulatory effects marked.

Either of these drugs used separately is not satisfactory. The vagomimetic action on the tracheobronchial tree is marked when hydergine is used alone, and the adreno-sympatheticomimetic action on the myocardium or respiration and on the basal metabolism is marked when diethazine is used alone.

Adrenolytic drugs include chlorpromazine (Largactil), which has a central and peripheral neuroplegic effect. Regitine and the di-hydrogenated derivatives of rye ergot (hydergine) cannot be used in its place.

Ganglion-blocking agents include tetra-ethyl-ammonium, procaine amide, methonium and arfonad.

Parasympatholytics, opposing the action of acetylcholine, include pethidine, atropine and antrenyl.

Antihistaminic derivatives of phenothiazine (promethazine, phenergan) or others like benadryl hold the precapillary sphincter closed and also contribute their interesting secondary effects, viz. hypnotic, anti-emetic, anticholinergic, (slight), analgesic and antipyretic.

The result of neuroplegia is as follows:

1. Endocrine inhibition—hypophyseal and suprarenal (chlorpromazine is adrenolytic but not noradrenolytic). Neuroplegia diminishes post-operative eosinopenia and diminishes or prevents the loss of ascorbic acid in the suprarenal glands.

2. Direct cellular action—diminishing cellular activities.

3. Neuroplegia plus hibernation produces a hypocoagulability attributed to sympathalase. This is seen with chlorpromazine alone (Huguenard[9]).

The drop of body temperature seen in neuroplegia is entirely due to the temperature of the surrounding air (Delorme[6]). It is concluded that massive doses of chlorpromazine do not significantly lower the temperature or oxygen consumption; it has an adverse effect on the natural defensive reactions unless supplemented by transfusion or by lowering of the metabolism (the lowering is minimal with the drug alone). Dundee[11] found in animals that deep anaesthesia, curarization or lytic cocktails were equally effective in facilitating hypothermia, and chlorpromazine alone apparently as effective as lytic cocktails in potentiating surface cooling.

Other Methods of Cooling

(*b*) *Surface Cooling*. This is effected by a variety of methods, the commonest of which is the immersion of the patient in an iced bath. Other methods are the use of refrigeration blankets, cold fans, etc. All of these must be combined with an anaesthetic and neuroplegic agent.

(*c*) *Vascular Cooling*. The arterio-venous method is simple. An artery is cannulated and the blood circulated through a cooling coil and returned to a vein. This, however, leads to undesirable side-effects, and veno-venous cooling is better. For this a pump is required.

(*d*) *Internal Cooling*—by circulating iced water through plastic bags in the stomach, or by pouring cold saline into the open thorax. These methods have not been used to any great extent.

Comparison of Methods of Cooling

1. *Extracorporeal venous cooling* is rapid. This method was devised by Delorme[6] in 1949 and by Boerema.[12] Later it was much used clinically by Brock and Ross.[1] These latter recommend rapid cooling and re-warming. They did not commence the cooling process until the chest had been opened, and then any cardiac irregularity could be rapidly dealt with. One advantage of venous cooling is that it allows of differential hypothermia, the cooled blood being returned to the proximal aorta and shunted mainly to the heart and brain. Thus the vital organs can be cooled rapidly to a sufficient degree to permit circulatory arrest for the allotted time. Re-warming then occurs equally rapidly. Whatever method of cooling is employed, the blood returning to the heart will be cooler than that of the rest of the body except the skin; hence the need for oesophageal temperatures, as giving a reasonably accurate index of the temperature of the heart itself.

The great disadvantage of this method is that cooling of the heart may occur too rapidly, which increases the risk of ventricular fibrillation, especially if instituted before thoracotomy has been done. Dangerous side-effects also arise from circulating the blood outside the body.

Hypothermia augmented by an assisted circulation of low or high flow (heart-lung machine) is much safer, prolonging the time of safe circulatory arrest. Cannulation of vessels will be necessary and when extra-corporeal venous cooling has been employed the stage will be set for assisted circulation. In the future perhaps this will prove to be the greatest advantage of this method of cooling.

Surface Cooling by bath immersion is simple, the equipment is readily obtained, and cooling can be well controlled. It is the method that has been most generally employed up till the present, particularly in the USA.

Helmsworth,[13] in a comparative study of hypothermia induction in dogs, found that surface cooling with iced water produced more profound haematological changes than extracorporeal cooling (Table I).

TABLE I. COMPARISON OF METHODS OF COOLING

	Extracorporeal circuit without cooling (90 min.)	*Water immersion to 23°C (60 min.)*	*Extrocorporeal cooling*
Haematocrit ..	15·8% increase	27·2% increase	20·4% increase
Platelet	9·6% decrease	80·7% decrease	24·8% decrease
Leucocyte ..	25·1% decrease	88·5% decrease	51·2% decrease
Total Eosinophil	13·2% decrease	85·0% decrease	33·1% decrease
Mean Corpusc. Hb. Content. ..	5·2% decrease	6·1% decrease	4·7% decrease

An advantage of the venous cooling method is that in the doubtful case cooling need not be commenced until a diagnosis to justify hypothermia is established at thoracotomy. However, in the majority of cases the decision to use hypothermia can be made beforehand. A disadvantage of extracorporeal venous cooling is the employment of a mechanical pump and the circulation of the blood through coils of plastic tubing which in themselves may lead to complications of blood clotting and destruction. After 300 experiments with dogs Delorme[6] concluded that cooling from 38° to 25°C by the vascular method, and re-warming, was innocuous *per se*.

Control of Temperature

The control of temperature is of the greatest importance because at too low a temperature there is a danger that cardiac irregularity will develop. Cooling is actively employed down to a temperature of about 31°C according to the end temperature desired. After active cooling has ceased, the body temperature continues to drop a further 2-4°C. This drop is more marked where surface cooling is employed, because the surface tissues are colder than those at a depth. In a fat patient this 'after drop' will be greater. Surface temperatures would then be misleading. Oesophageal or rectal temperatures are awkward to take with the conventional thermometer. An oesophageal thermocouple with galvanometric recordings, though it entails special equipment, is desirable both for safety and convenience.

The rate of cooling is also important; too rapid cooling is dangerous. Children tolerate hypothermia better than adults and can have their temperature lowered faster and lower. It needs

30-40 minutes to reduce an infant's temperature from 37° to 31°C (about 1°C for each 5 minutes). An adult will cool less rapidly and will probably have to be immersed in the bath for fully an hour. Refrigeration blankets, although less cumbersome and more aesthetic than bath immersion, require 2-3 hours to achieve the same effect. Constant electrocardiographic monitoring is necessary throughout this period. The best instrument for this is the cathode-ray oscillograph.

The re-warming period is just as dangerous and important. It should take about as long as the cooling. When the oesophageal temperature has reached 35°C the patient will be sufficiently 'round' for the intratracheal tube to be removed and the anaesthetist to leave. Spontaneous respiration should then be sufficient. It is to be realized, however, that apart from the anaesthetic used for induction and a long-acting muscle paralysant, no anaesthetic agent is needed throughout the major part of this procedure. The lowered temperature is sufficient anaesthetic for all surgical procedures. However, there is an individual variation in the patient's response and in a few cases a little anaesthetic will be required.

Re-warming is done in various ways. If left alone at room temperature the patient will gradually warm up once the effect of the drugs has worn off. Glen[5] cooled dogs to 25°C; without any artificial respiration or supervision they were returned to their kennels, and all were well the next morning. However, it is generally desirable to start warming the patient as soon as the need for hypothermia is over. This stage is reached when the definitive procedure is completed. This re-warming is commenced while the operation is still in progress, and is effected by means of electrically warmed blankets or, better still, specialy constructed rubber blankets through which warm water is circulated. These are put into place at the commencement of the operation. Other methods are to use a diathermy coil or radio-frequency (Bigelow[16]), which has the advantage of warming the deeper tissues. Warm saline in the chest before closure will also help. At the end of the operation the patient's temperature may hardly have started to rise. The warm-blanket treatment can be continued or the patient may be immersed in a warm bath until the temperature has risen to 35°C. In some of our patients consciousness has returned at a lower temperature; one child was returned to the ward while the oesophageal temperature was 33°C.

METHODS OF AUGMENTING HYPOTHERMIA

Various authors indicate what they think is the safe period of venous occlusion at different levels of hypothermia. The temperatures most surgeons prefer are 28°-31°C. Below this the risks of ventricular fibrillation are greatly increased. At 28°C the circulation can safely be occluded for 10 minutes without fear of ischaemic damage. Nevertheless, during circulatory occlusion the heart still contracts and is in need of a proportionately greater amount of oxygen. The following methods are used to supplement hypothermia.

(*a*) *Controlled Asystole*

Controlled asystole can readily be produced by perfusing acetylcholine or prostigmine through the coronary circulation. This prostigmine effect will be eliminated as soon as the blood-flow through the coronary arteries washes it out. Potassium chloride and magnesium sulphate can be similarly used (Young[18]). However, the cold heart frequently fibrillates after release from standstill induced by potassium.[15] Prostigmine slows the heart rate at the critical post-standstill period (Sealey[16]).

Swan[17] found defibrillation easier if potassium chloride was injected into the coronary circulation. A somewhat conflicting opinion is expressed by Reberi,[19] who found the standstill produced by perfusing the coronary arteries with potassium chloride difficult to convert to normal rhythm. Perfusion with calcium chloride at this time often produced fibrillation. Because of these conflicting views we have not used potassium chloride to produce cardiac arrest. Instead we have used prostigmine wherever necessary. Jordan[19] has found potassium chloride with glucose and insulin to be safe and effective.

(*b*) *Differential Hypothermia*

By circulating oxygenated and refrigerated venous blood in a gas-dispersion pump oxygenator, Gollam[20] was able to achieve survival of dogs after cardiac arrest for 1 hour at 0°C. In these experiments, in 40 minutes of blood cooling the temperature inside the right heart was lowered to 5°C and in 15 minutes of blood warming it was raised to 27°C. Differential hypothermia between upper and lower parts of the body reached 20°C. The last heart contraction was noted at 13°C. On re-warming, the pump was turned off at 28°C. Gollam gave quinidine and had no ventricular fibrillation, no anoxia of the central nervous system, heart or viscera, no CO_2 retention and no air embolus in asystole. Cortisone was used to prevent the high mortality that occurs after short circulatory cessation below 20°C. He gave his animals 100 mg. of cortisone acetate intramuscularly on the day before the operation, 100 mg. of hydrocortisone intravenously before cooling, and 50 mg. intramuscularly on that and the next 3 nights. At these low temperatures a low systemic blood pressure is adequate (20-30 mm. Hg), but by the time 21°C is reached a higher pressure is required and this is achieved by an intravenous drip of 20 mg. of L-arterenal (nor-adrenaline) in 500 c.c. of plasma expander (continued slowly throughout the re-warming period, 4-5 hours). Gentle cardiac massage is also necessary until 22-23°C is attained.

Delorme[6] produced differential hypothermia of 7-10°C by perfusing the pericardial sac with iced saline after the rest of the body temperature had reached 25°C and the inflow had been occluded.

In the presence of a septal defect, differential cooling may occur, causing the heart and lungs to be cooler than the rest of the body (Brock[1]).

(*c*) *Low-Flow Extra-Corporeal Circulation*

Battezzati[21] reported that 22 out of 26 dogs survived complete cardiac occlusion and right ventriculotomy for 13 to 56 minutes. He cannulated the vena cavae and cooled the blood through coils in ice and oxygenated it before returning it to the carotid artery. He contended that 40-50 c.c. per minute was sufficient for a dog at 28°C, 60-70 c.c. at 32°C, and 100 c.c. at normal temperature. The blood was heparinized and he advocated the use of an equal dose of protamine sulphate to counteract the effect of the heparin.

Using a low-flow extra-corporeal circulation, Sealey[16] has kept the cold heart in standstill and ischaemic for 31 minutes in a dog and for 17 minutes in a man. He considers cardiac standstill to be safe for 13-30 minutes at 30°-32°C, and that flow rates of as little as 15 c.c. per kg. per minute are probably safe for 10-15 minutes, 30 c.c. per kg. for 15-20 minutes and 40 c.c. per kg. for 20-30 minutes. He has carried a 23 kg. patient through 21 minutes perfusion at 26 c.c. per kg. per minute.

Swan[17] found a flow rate of 50-85 c.c. per minute with pressure range of 25-85 mm. Hg adequate in dogs. He used plastic bags for oxygenation. For anticoagulation he used 0·5 mg. of heparin per kg. through the venous outflow catheter. To neutralize this, a 0·05% solution of protamine sulphate in 0·85% sodium chloride was given by slow drip. The estimated protamine dosage was mg. for mg. of heparin, including that given in the donor blood. The clotting time returned to normal ½ hour after starting protamine.

Ross[22] also advocated relief of the heart's work by the use of the heart-lung machine.

(*d*) *Coronary Perfusion*

Bahnson[23] used the ordinary intravenous blood-transfusion apparatus suspended from the ceiling to provide for coronary perfusion. For this purpose oxygenated heparinized blood was collected from donors by heating the arm in a bath for 15 minutes and collecting venous blood in the usual way.

Bailey[24] found that arterial transfusion to maintain high pressure in the aortic arch reduced the chance of coronary air embolus. For transfusion he advocated the use of red corpuscles suspended in gelatin-Ringer solution. The use of arterial transfusion prolongs the period at any given temperature during which the circulation can be clamped off. He has closed the circulation for 22 minutes for the repair of a septal defect.

Gollam[20] had only one dog survive 12·0°C, but by circulating oxygenated blood had one survive 0°C.

Shumway[25] used coronary perfusion to protect the heart. He used a Sigmamotor pump. The right heart was aspirated through either right cardiotomy or through an incision in the pulmonary artery. This blood was re-circulated. Thus only a reservoir of 500 c.c. of blood was needed. At 25°C he found that a flow rate of 50-85 c.c. per minute with pressure range of 25-85 mm. Hg was adequate. To prevent clotting he used 20 mg. of heparin in 500 c.c. No neurological defect was observed in any dog with circu-

latory occlusion for 20 minutes at 25°C, but it did occur in 2 out of 6 occluded for 30 minutes. He observed no ventricular fibrillation in 7 dogs in which the coronaries were perfused with oxygenated blood. He mentions 2 clinical cases of atrial septal defect in which unexplained post-operative bleeding took place. The flow rate was 100 c.c. per minute. Both fibrillated but this was easily reversed. On perfusion of the coronary arteries of 10 dogs with venous blood, all survived 20 minutes of occlusion; no post-perfusive transfusions were necessary, but two developed ventricular fibrillation. The flow rate was 50 c.c. per minute (a reservoir of 1,000 c.c. of heparinized blood was sufficient). The coronary drainage was discarded, and the oxygen content averaged 4·4 vols.% less than the perfusate, indicating that the myocardium extracted oxygen as readily from the venous blood as from arterial blood.

INDICATIONS FOR HYPOTHERMIA

Shock

Controlled hypothermia has been used as an aid in the treatment of severe shock and prophylactically for poor general condition before major procedures. Van Krankenanstalen[26] reported its use in over 70 such cases. Internal temperatures of 34-35°C should be maintained for 48-100 hours. He advocated this procedure in severe injuries, including head injuries, and in intoxication.

Controlled hypothermia for this purpose, particularly that induced by the administration of drugs, has received wide acclaim in France (artificial hibernation of Laborit).

Recently Lewis[27] advocated a 'technique for total body cooling of the febrile gravely ill patient'. The first case he had was one in whom an operation for atrial septal defect had just been completed. After the operation the patient could not maintain her condition. There was immediate improvement on cooling. Since then this method has been used on 24 other gravely ill patients.

Cardio-vascular Surgery

It is in this field that the greatest application of hypothermia will be made in this country at present. Later, when a safe method of maintaining an artificial circulation with pump oxygenator has been established*, the scope of operations performed under hypothermia will be restricted. At present it is the only real aid we have that will enable us to work on a vital organ in a completely bloodless field with the added safety of prevention of haemorrhage and lessened possibility of tissue death. At present the operations which we feel should be performed under hypothermia are the following*:

1. Those requiring cardiac arrest
   - (*a*) Open cardiotomy: (i) Atrial septal defect, (ii) intra-atrial lesions, (iii) mitral incompetence and recurrent mitral stenosis, (iv) ventricular septal defect, (v) right ventricular infundibular stenosis.
   - (*b*) Opening of aorta and pulmonary vessels: (1) Aortic stenosis, (ii) pulmonary stenosis, (iii) aortic pulmonary fistula.
   - (*c*) Other operations on the proximal aorta.
2. Those not requiring cardiac arrest
   - (*a*) Aneurysms of the distal thoracic and upper abdominal aorta,
   - (*b*) Various shunt operations on severely cyanotic patients (Blalock's and Pott's operations),
   - (*c*) other operations.

1. *Operations Requiring Cardiac Arrest*

(*a*) *Open Cardiotomy.* Lewis[9] is credited with first using hypothermia in the closure of an atrial septal defect. He considered the advantages of this method over the heart-lung machine were: (1) it is completely bloodless and there is no coronary flow; (2) the chance of air embolus is reduced if the inflow and outflow are shut; (3) it is simple. At the time of writing he had operated upon 11 atrial septal defects. It is interesting to note that as late as 1957 Lillehei,[28] who had performed over 200 operations using the pump oxygenator, recommended that atrial septal defect of the osteum secundum type be treated under hypothermia.

By 1957 Holmes Sellors[29] had closed 40 cases of atrial septal defect under hypothermia. Bigelow[30] advocated the use of hypothermia in adult atrial septal defects and in serious-risk cases, e.g. mitral stenosis with right ventricular failure and the cyanotic group of congenital heart diseases.

On the other hand Gross[31] was until recently still using his 'well technique' for the closure of atrial septal defect, while Kirklin[32] even though he has an elaborate and smoothly running pump-oxygenator, still repairs atrial septal defects by Gross's technique.

Our present experience of open cardiotomy has been in 2 cases of right ventricular infundibular stenosis and in 2 cases of atrial septal defect. There is a place for this technique in the removal of interatrial tumours, for the repair of mitral incompetence, and for the relief of mitral stenosis where previous attempts have failed. Ventricular septal defects are not commonly dealt with under hypothermia, for the time available for circulatory occlusion is not really adequate, although with differential hypothermia and coronary perfusion successful repair has been reported (Bahnson[23]).

(*b*) *Pulmonary and Aortic Stenosis.* The approach is retrograde through the opened aorta or pulmonary artery during circulatory arrest.

Swan,[33] who has had extensive personal experience of hypothermic operations, quotes 52 cases of the open relief of pulmonary stenosis without a death. By 1956 he had reported the use of hypothermia in 130 congenital cardiac cases.

In Johannesburg this approach has been used in 4 cases each of congenital aortic stenosis and pulmonary valvular stenosis and in 2 cases of common truncus arteriosus.

(*c*) *Other Operations.* Hypothermia is ideally suited to the repair of aneurysms of the aortic arch or innominate artery where cessation of circulation to the brain may be necessary.

2. *Operations not requiring Cardiac Arrest*

Thornton,[5] working in Professor Rob's clinic, has described 71 cases without death. Mostly these were the placement of aortic grafts in the descending aorta close to the renal vessels.

We have performed shunt operations (Blalock's and Pott's) in 4 severe cyanotic children, 2 for a Fallot's tetralogy and 2 for congenital tricuspid stenosis. We have also used hypothermia for the ligation of patent ductus in 2 children with pulmonary hypertension.

Uglov[24] has used hypothermia in a variety of other intrathoracic procedures, including pulmonary resection.

COMPLICATIONS OF HYPOTHERMIA

1. *Ventricular Fibrillation* (VF)

This complication is common in hypothermia below 28°C. It is produced in the main by the lowered temperatures, rough cardiac manipulation and coronary air embolus.

Shumway[25] reported it in one-third of his clinical cases, but found defibrillation successful in all but one case. Swan[17] reported VF in 100% of dogs not protected by prostigmine or acetylcholine in which right ventriculotomy was done, all cooled to 25°C. He recommended coronary perfusion with Sigmamotor pump at a coronary pressure of about 70 mm. Hg to prevent air embolism. He pointed out the danger of using citrate, which is toxic to the hypothermic myocardium. Cookson[35] reported that with hyperventilation at 20-25°C VF resulted in only 8% of cases. During cooling and hyperventilation, serum-potassium levels fell. He found defibrillation easier if potassium chloride was injected into the coronary circulation (air embolus is unlikely to occur if the heart is arrested). Reberi[18] and Young[16] expressed a contrary opinion. Delorme[6] thought VF was due to lowered potassium in the cells possibly due to sodium entering the cell or potassium leaving.

Battezzati[21] used extracorporeal circulation to lessen the VF of 26 dogs, of which 22 survived right ventriculotomy plus right and left auriculotomy for 13 to 56 minutes (venous cooling). He advocated the use of a pulsating flow.

Khalil[36] used internal cooling (gastric bag) and in 20 rabbits cooled to 20°C had no VF. The process took 2½ hours. He safely cooled one hyperpyrexial child to 23-24°C.

Brock[1] gave no preliminary intravenous procaine injection, and suggested rapid cooling and re-warming (venous cooling) to prevent VF. He also[37] recommended maintaining adequate coronary flow by (*a*) simple veno-arterial pump circuit, (*b*) clamping the aorta beyond the left subclavian, and (*c*) the heart-lung machine. To manage a VF he considers it is necessary first, if possible, to repair the defect and then restore myocardial tone by injecting 5-10 c.c. of 1/10,000 adrenaline in normal saline, or the same quantity of 1% calcium chloride, into the left ventricle; also to cross-clamp the aorta. A noradrenalin drip increases perfusion pressure and slows the heart. Factors increasing the heart rate

* We have had a Gross-type disc oxygenator in use since August 1958 and anticipate that this aid will be used in operations on most of the conditions listed under this heading.

must be avoided, such as unnecessary trauma, heat from the operating lamp and warm instruments; prostigmine may be used to slow the heart.

Thornton[5] re-warmed slowly, no faster than 1-1·5°C an hour. He is emphatic that one should release inflow clamps slowly to avoid VF (endogenous epinephrine stored up in the inferior vena cava acts on the sensitized myocardium (Covino[38]).

Glen[6] cooled dogs with the aid of an arteriovenous extracorporeal circuit. VF only occurred below 23°C.

Ross[22] has given up chemicals like prostigmine and advocates slow release of the vena cava after arrest. He has experienced no difficulty in defibrillating the hypothermic heart provided massage produced adequate tone. Venous pooling occurs and his aim is to under-transfuse and keep respiratory acidosis at a minimum.

Covino[39] advocated using ambonestyl intravenously or by mouth and artificial ventilation. To show this he performed

TABLE II. VENTRICULAR FIBRILLATION AFTER COOLING

Cooling without artificial ventilation	80% VF
Cooling with artificial ventilation	40% VF
Cooling with ambonestyl I.V. 40 mg./kg.	30% VF
Cooling with ambonestyl and artificial ventilation ..	0% VF

controlled experiments at 25°C (Table II). It was his opinion that the circulation could be stopped for 15 minutes at 20°C.

Radigan[39] infiltrated the superior atrio-caval junction (S.A. node) with procaine. Without this 18 out of 20 dogs developed VF. With procaine there was no VF. He suggested that there is a neural causal component. He has used this method in 11 patients without VF. We have used it in all but one of the cases where circulatory arrest was applied. Reberi[18] also used this method (1% procaine), and reported that the P wave disappeared or decreased, the pulse rate slowed, and the blood pressure fell moderately, these changes lasting 12-27 minutes at 25-28°C. All 12 controls fibrillated and the 11 treated did not. At lower temperatures only 1 of 16 treated animals fibrillated. Reberi listed the following procedures in dealing with established ventricular fibrillation. (1) If the myocardium is weak during fibrillation give intracardiac epinephrine (a few c.c. of 1 in 20,000). (2) Give the procaine injection both before and after. (3) Block the sympathetic supply to the heart either with arfonad or by stellate-ganglion block (upper 3 thoracic). (4) If the fibrillation is due to coronary air embolus, massage the heart to express the air from the vessels. (5) Defibrillate with high voltages (greater than 110 volts).

Kyle[40] in comparing the effectiveness of calcium chloride and electric shock in defibrillating hearts of hypothermic dogs stated: (1) Serial electric shocks after 4 minutes of fibrillation produced 100% defibrillation with effective heart beat in 100% of cases. (2) Calcium chloride after 4 minutes of fibrillation produced 100% defibrillation but only 57% effective beat. (3) Calcium chloride after 1½ to 2 minutes produced 100% defibrillation but only 87% effective beat.

Cookson[41] gave ACTH beforehand, injected procaine into the pericardium after unclamping the venae cavae, and injected benedaine (adrenolyte), 0·8 mg. per kg., into the right ventricle. VF has been much lessened since using this method. He cooled to 26°C in 2½-3½ hours.

Bigelow[42] tried an electrical pacemaker to control the heart beat. The prospects of such an apparatus are good.

Many experimenters have warned against the danger of transfusing cold blood. This, if given rapidly, sometimes causes a serious drop in the temperature of the heart. For this reason, and because there is a tendency for blood to pool on the venous side of the circulation, intra-arterial transfusions are often given.

Lewis[8] injected butocaine into the pericardium (4-8 c.c. of 10%) and passed tapes around both lung roots to occlude the pulmonary veins; he used procaine hydrochloride before and during cooling and VF did not occur.

2. *Tissue Anoxia*

There are conflicting reports on whether cooling alone will produce tissue anoxia. Probably artificial respiration will prevent the development of tissue necrosis.

Sarajas[43] found myocardial necrosis in animals cooled from 27·5°C to 20°C and kept thus for 1-4 hours. Glen[6] interrupted the cerebral circulation for 30 minutes in animals at 25°C, and all recovered normally.

Virtue[17] found that at 28°C 50% of cases showed auricular fibrillation of ECG. A disturbing feature was the high take-off of the S.T. segment half way down the R.S. segment indicating myocardial damage or hypoxia. This frequently preceded VF.

Ross[22] considered the main hazard in 70 patients with circulatory arrest for 5-10 minutes to be cerebral anoxia. There were 5 recoveries out of 8 in whom cerebral circulation was absent or poor for more than 10-12 minutes. He stated that increased notching or slurring of the QRS complex often preceded VF.

Cahn[44] stated that at 30°C the ECG showed curves similar to those of hypometabolism. He thought that prolonged anoxia resulted in myocardial failure but that cardiac resuscitation could be obtained with an injection of adenosine triphosphate plus cytochrome.

Edwards[45] showed that the oxygen consumption of the hypothermic heart muscles was reduced but there was no discrepancy between myocardial oxygen supply and demand. He did not think hypoxia was a factor in the production of VF. Bailey[24] clamped off the circulation in dogs for 40 minutes at 16°C without detectable brain damage. Delorme[6] found no renal changes (functional or anatomical) after clamping the kidney pedicle for 4 hours at 25°C; similarly with the liver. There were no nerve-cell changes after cerebral ischaemia for 1 hour at 25°C. Huguenard[9] found no histological lesion in hibernation, except at temperatures below 27°C.

The general conclusion from the available data is that within the temperature ranges used for hypothermia operations tissue damage is not likely to result from the lowered temperature alone.

Other Complications

3. *Air Embolism.* The greatest danger of air embolism is to the coronary circulation. If it occurs, manual systole must be used with the aorta clamped to propel the air bubbles out of the vessel. Because of the danger of coronary air embolus, some do not advocate open left cardiotomy. The danger could be diminished by cross-clamping the aorta at the level of the coronary arteries. When operating on septal defects one must be careful not to suck the blood out of the left side, and it is important to fill the chambers with saline before finally closing the heart. Cerebral air embolism has not been found troublesome. A veno-arterial shunt to maintain sufficient pressure to keep the aortic valves closed also helps. The use of asystole has eliminated the possibility of air being propelled along the coronary arteries while the heart is open.

4. *Acidosis.* Scurr[46] has drawn attention to the synergism between carbon dioxide and the neuromuscular blocking agents. He therefore warned against respiratory acidosis. It is thought that ventricular fibrillation is more likely to occur if acidosis has developed.

5. *Bleeding.* Swan[23] stated that the major cause of death in 105 cases was disturbance of the clotting mechanism. Shumway[25] could find very little cause experimentally for the post-operative bleeding (the clotting times, prothrombin index, plasma, haemoglobin and platelets were normal). Haemorrhage may be due to liver anoxia and citrate poisoning with severe calcium depletion. Osler Abbot[47] suggested that the calcium should be removed from the blood by a resin membrane so as to prevent its clotting, and thus obviate the need to use citrate or heparin.

6. *Pneumonia* has been reported in a number of patients who have been subjected to hypothermia.

7. *Curare Retention.* Thornton[5] has given a warning about the tendency to recurrence of neuromuscular block which previously has been reversed by neostigmine. Perhaps curare may persist in some depot during hypothermia, or if renal circulation has been interrupted there might perhaps be delay of curare excretion. However, Bigelow[48] attributed this effect to CO_2 build-up.

8. *Temperature Regulation Defect.* In certain cerebral conditions where there is a disturbance of the heat regulating centre, the 'after drop' in temperature may be excessive and difficult to overcome.

9. *Local Fat Necrosis.* Delorme[6] reported fat necrosis as having occurred after surface cooling in infants.

10. *Burning during Re-warming.* This has occurred with the use of diathermy coils.

OPTIMUM TEMPERATURES

On this point there is no unanimity of opinion, although most workers consider it inadvisable to reduce the internal temperature below 28°C.

Cookson[41] cooled to 26°C and Ciocatto[49] to 29°-28° for bloodless

open heart surgery. Swan[50] recommended reducing the temperature to 26°C, recorded by rectal thermocouple, for closure of atrial septal defect. By stopping the cooling process at 34°C and allowing the patient's temperature to drift to 30-31°C, and using extracorporeal blood oxygenation, he[17] has maintained safe cardiac asystole for 17 minutes in man. He therefore contended that with hypothermia of 30°-32°C the safe period of standstill with ischaemia is between 15 and 30 minutes. (He has had no difficulty with post-operative bleeding.)

Gollam,[20] using pump oxygenation, had dogs survive cardiac arrest for 1 hour at a differential hypothermia at 0°C; at 15°C 2 dogs died after 15 minutes' inflow occlusion and 2 were not quite normal after 12 minutes occlusion. However, Shumway[25] found no neurological defects in any dog after circulatory occlusion for 20 minutes at 25°C, but he did find neurological defects in 2 out of 6 dogs occluded for 30 minutes. Lewis[8] operated upon 11 cases of atrial septal defect at 29°C. Holmes Sellors[51, 29] has performed the closure of atrial septal defect on 40 cases with one death; he cooled the patients to 29-30°C. Bailey[24] considered that the optimum temperature in severely cyanotic infants was 23·3-26·1°C and for surgery to the great vessels involving interruption of circulation to vital centres for prolonged periods 21·1-24·4°C; but that in adults the temperature should not be reduced below 26·1°C. Kaplan[4] reported 22 patients between the ages of 18 days and 4 years in whom the rectal temperature was reduced to 24°C.

We have tried to keep the oesophageal temperature above 29°C but in several it has fallen below this and in 2 patients reached 28·1°C without any ill effect.

HISTORICAL

In 1875 Reincke[52] wrote an article on 'Observations on accidental hypothermia in man'. In 1938 Temple Fay[53] described attempts to control malignant disease by hypothermia produced by surface cooling. This stimulated other workers and investigations into the effects of climatic cold followed.

As an aid to cardiac surgery hypothermia is directly the result of the experimental work done by Bigelow[42] of Toronto, in 1950. This was put to the clinical test by Cookson and Bailey (1952)[41] Lewis and Taufic[54] (1953), Swan and Zeavin[50] (1954), Bailey and Cookson[24] (1954). Bigelow[55] and Boerema[12] are credited with the conception of hypothermia to allow cessation of the circulation.

Lewis and Varco,[54] 1952, were the first people to close an atrial septal defect under hypothermia and direct vision. Cooling by the extracorporeal (arterio-venous) circuit was originated by Delorme[6] and Boerema[12] in 1951. Venous cooling was originated by Ross in 1954.

Hypothermia in South Africa

Many of the cardiovascular procedures that have been undertaken in this country under hypothermia would be more adequately performed with the use of an artificial oxygenator (heart-lung machine).

To date I have had experience with 24 clinical cases operated upon under hypothermia. These have included 2 cases of open right cardiotomy, where the right ventricle has been opened and an infundibular obstruction resected under direct vision, 2 cases of an atrial septal defect, and 4 cases each of pulmonary valvular stenosis and congenital aortic stenosis with retrograde approach to the valves *via* an incision in the respective arteries. All these 12 cases necessitated complete circulatory occlusion for 5-7 minutes. The remainder of the operations have been on the great vessels outside the heart, in only 2 of which was circulatory arrest needed.

The use of hypothermia is simple and safer than the artificial oxygenator and for some years yet it is likely to be employed in a variety of conditions. Against this is the disadvantage of limited time in which to work and the inability therefore to repair the more complicated intracardiac defects.

A review of the individual cases will be presented in the future. In the meantime we and the anaesthetists concerned have been fully convinced of the safety of the procedure as at present practised here.

This paper would not be complete without paying tribute to those anaesthetists who have so courageously undertaken this pioneering work in Johannesburg. I head the list with the name of Dr. F. W. Roberts, who administered the anaesthetic to the first group of patients, and add those of Drs. C. H. van Hasselt, K. B. Meaker, J. R. Duffield, H. Ginsberg, C. Frost and D. Jeffes. Also I must thank the theatre staff at the Children's Hospital, Johannesburg, who willingly coped with many teething difficulties presented by hypothermia, and Sister Tickner in particular. Dr. C. H. Wyndham and his team from the Chamber of Mines Research Laboratories devoted much time to galvanometric temperature recordings in our first four cases, and without their help this work may well have not commenced. I thank Dr. L. Braudo for his constant encouragement and help.

REFERENCES

1. Brock, R. C. and Ross, D. N. (1955): Guy's Hosp. Rep., **104**, 99.
2. Thornton, H. L. (1957): Brit. Med. J., **1**, 253.
3. Muller, W. H. (1954): Discussion on Bailey,[24] J. Thor. Surg., **27**, 1.
4. Kaplan, S. (1954): Discussion, *Ibid.*, **27**, 1.
5. Glen, A. (1954): S. Afr. Med. J., **28**, 38.
6. Delorme, F. J. (1955): Brit. Med. Bull., **11**, 221.
7. Holmdahl, M. (1955): Proc. World Congress Anaesth.
8. Lewis, F. J. (1954): Surgery, **36**, 3.
9. Huguenard, P. (1955): Proc. World Congress Anaesth.
10. Bimar, J. G. (1955) *Ibid.*
11. Dundee, J. W. (1953): Brit. Med. J., **2**, 1,244.
12. Boerema, I. A. (1951): Arch. Chir. Med., **2**, 25.
13. Helmsworth, J. A. (1956): Arch. Surg., **73**, 3.
14. Bigelow, W. G. (1952): Canad. J. Med. Sci., **30**, 1,857.
15. Young, W. E. and Sealey, W. C. (1956): J. Thorac. Surg., **32**, 604.
16. Sealey, W. C. (1957): Surg., Gynec. Obstet., **104**, 4.
17. Swan, H. and Virtue, R. V. (1953): J. Amer. Med. Assoc., **153**, 12.
18. Reberi, A. and Shumaker, H. B. (1955): Surgery, **38**, 5.
19. Jordan, P. (1957): Surg., Gynec. Obstet., **105**, 615.
20. Gollam, F. (1955): J. Thorac. Surg., **30**, 5.
21. Battezzati, M. (1955): Proc. World Congress Anaesth.
22. Discussion on Hypothermia (1957): Proc. Roy. Soc. Med., **50**, 2.
23. Bahnson, H. T. *et al.* (1957): Surgery, **42**, 76.
24. Bailey, C. (1954): J. Thorac. Surg., **27**, 1.
25. Shumway, N. E. (1955): *Ibid.*, **30**, 5.
26. Von Krankenanstalten, E. G. (1955): Proc. World Congress Anaesth.
27. Lewis, F. H. (1956): Surgery, **40**, 3.
28. Lillehei, C. W. (1956): *Cardiovascular Surgery Panel Discussion*, Second World Congress of Cardiology—ed. H. B. Tausig New York: Hoeber-Harper.
29. Sellors, T. H. (1957): Lancet, **1**, 1255.
30. Bigelow, W. G. (1954): Discussion, Surgery, **36**, 3.
31. Gross, R. (1954): Discussion, *Ibid.*, **36**, 3.
32. Kirklin, W. (1957): Discussion, Arch. Surg., **75**, 2.
33. Swan, H. (1956): Discussion, *Ibid.*, **73**, 3.
34. Uglov, F. G. (1955): Proc. World Congress Anaesth.
35. Cookson, B. A. (1954): Ann. Surg., **139**, 430.
36. Khalil, L. (1957): Lancet, **1**, 185.
37. Milstein, B. B. and Brock, R. C. (1954): Guy's Hosp. Rep., **103**, 213.
38. Covino, B. G. (1956): Surgery, **40**, 3.
39. Radigan, L. R. (1956): *Ibid.*, **40**, 3.
40. Kyle, R. H. and Kirby, C. J. (1957): Arch. Surg., **74**, 1.
41. Cookson, B.A. and Bailey, C. (1952): Dis. Chest., **22**, 3.
42. Bigelow, W. G. (1950): Amer. J. Physiol., **160**, 125.
43. Sarajas, H. S. S. (1956): Amer. Heart J., **52**, 6.
44. Cahn, (1955): Proc. World Congress Anaesth.
45. Edwards, W. S. (1956): Ann. Surg. **139**, 275.
46. Scurr, C. F. (1954): Brit. Med. J., **1**, 562.
47. Abbot, O. (1955): J. Thorac. Surg., **30**, 5.
48. Fairly, H. B., Waddell, W. G. and Bigelow, W. G. (1957): Brit. J. Anaesth **29**, 310.
49. Ciocatto, E. (1955): Proc. World Congress Anaesth.
50. Swan, H. (1953): Ann. Surg., **138**, 3.
51. Sellors, T. H. (1957): Lancet, **1**, 443.
52. Reincke, J. (1875): Dtsch. Arch. klin. Med., **16**, 11.
53. Fay, T. (1938): Surg., Gynec. Obstet., **66**, 512.
54. Lewis, F. J. and Taufic, M. (1953): Surgery, **33**, 52.
55. Bigelow, W. G. (1950): Ann. Surg., **132**, 531.

Northern Areas Division (Cape Western Branch, M.A.S.A.). The Annual General Meeting of this Division will be held in the Council Chamber, Klosser Street, Parow, Cape, on Thursday 27 November at 8.15 p.m. The telephone number of the venue will be 98-5223.

Afdeling Noordelike Gebiede (Tak Wes-Kaapland, M.V.S.A.). Die Algemene Jaarvergadering van hierdie Afdeling sal op Donderdag 27 November om 8.15 nm. in die Raadsaal, Klosserstraat, Parow, Kaap, plaasvind. Die telefoonnommer by die vergaderplek is 98-5223.

ADAPTATION, COMPLEMENT FIXATION, AND RECOMBINATION OF TYPES 1 AND 2 POLIOMYELITIS VIRUSES*

G. SELZER, M.B., CH.B., *C.S.I.R. and U.C.T. Virus Research Unit, Cape Town*

Adaptation of Type-2 Poliovirus to Suckling Mice

Type-2 poliovirus was adapted to suckling mice in order to have a readily available source of material of high titre. The virus used was the MEF_1 type-2 poliovirus. This virus was inoculated into several litters of albino mice aged 4 days. Four days later the brains and cords of 6 mice were harvested at random, pooled and inoculated into a further group of sucklings. Ten such mouse-to-mouse passages failed to adapt the virus to sucklings.

In a further attempt the sucklings were given a double inoculation, receiving 0·02 ml. of virus on one side of the brain followed by a similar dose on the other side 2 hours later. Passages were carried out as before at 4-day intervals. Presently a few sucklings became paralysed. The number gradually increased until by the 17th passage 100% of infected mice became paralysed on or before the 4th day after inoculation. By the 29th passage 100% were paralysed on the 2nd day.

The virus reached the maximum concentrated in the brain and cord immediately before the onset of paralysis and persisting for 2 days thereafter.

The Soluble Antigen of the MEF_1 Poliovirus

By ultracentrifugation of emulsions of brains from suckling mice infected with the MEF_1 strain of poliovirus, infective virus was separated from a non-infective soluble antigen. The soluble antigen which remained in the supernatant fluid was serologically specific and was responsible for most of the complement fixation shown by such brain emulsions.

This soluble antigen was heat-stable and resisted treatment with organic lipoid solvents such as ether, $CHCl_3$ and acetone. Inoculation of soluble antigen into adult mice did not stimulate the production of neutralizing antibodies.

A Comparison of Complement-fixation titres in Poliomyelitis-infected Tissue-culture Fluids and Mouse Brains

The complement-fixing titres of poliomyelitis-infected tissue-culture fluids is persistently low despite the high infectivity titres. MEF_1-infected suckling-mouse brains have 10 times the complement-fixing titre, yet the infectivity titre is less than that of the MEF_1-infected tissue-culture fluids.

Several experiments were carried out to show that MEF_1-infected tissue-culture fluids do not contain any soluble antigen. This appears to be the explanation for the low complement-fixation titres obtained.

Adaptation of Mahoney Type-1 Poliomyelitis Virus to Suckling Mice

Attempts to adapt the type-1 poliovirus to suckling mice by the methods employed for the adaptation of the MEF_1 type-2 strain proved unsuccessful.

A growth curve of the Mahoney virus in the brains of suckling mice revealed that the virus undergoes two 'cycles' of multiplication—the first within 48 hours after intracerebral inoculation, and the second approximately 10 days later. The former is not associated with paralysis, whilst the latter usually is.

By to-and-fro passages in suckling-mouse brains and monkey kidney-tissue culture there was a gradual increase in the amount of virus obtained during the early multiplication cycle in suckling-mouse brains and associated with this was the occurrence of early paralysis in 80% or more of these mice. The virus capable of causing early paralysis in infant mice was gradually lost by direct mouse-to-mouse passage.

The occurrence of two variants is postulated, one of these apparently non-paralytic in its properties.

At the 7th to-and-fro passage between mouse and tissue culture, 58% of suckling mice were paralysed within 4 days after inoculation. The pooled brains and cords of these mice were inoculated into 100 suckling mice. Only 2 mice were paralysed within 4 days, one of these within 24 hours. It was from the latter that the final mouse-to-mouse adaptation was obtained. This virus, now in its 30th passage in suckling mice produces paralysis in almost 100% of mice within 2 days.

The next step will be to produce an antiserum to the Mahoney suckling-mouse-adapted virus test for a soluble antigen and, if present, compare it with that of the MEF_1 virus. Paired sera of recently infected patients will also be tested for complement-fixing antibodies.

Recombination of Types 1 and 2 Polioviruses

Attempts were made to produce a virus which had some of the properties of both type-1 poliovirus and type-2 poliovirus. For this the suckling-mouse-adapted strains of Mahoney type-1 and MEF_1 type-2 viruses were used. The Mahoney had an incubation period of 48 hours, whilst that of the MEF_1 was shorter, viz. 24 hours. Both viruses were concentrated, that of the Mahoney to a greater degree. Varying mixtures of the two were inoculated into groups of suckling mice, and the brain and cord of each paralysed mouse harvested separately. The virus obtained from each mouse was characterized by neutralization tests using a type-1 and type-2 monkey immune serum obtained from the Poliomyelitis Research Institute, Johannesburg.

The majority of mice contained both viruses in varying proportions; some contained only Mahoney virus, but from one mouse a virus was obtained (by limit dilution techniques) which was neutralized by both antisera, suggesting that one was dealing with a recombinant. The one snag is that although this virus is neutralized by the type-1 monkey antiserum, it is not neutralized by the Mahoney type-1 mouse antiserum. The latter is a poor serum, and this may explain the poor result, but the matter still needs considerable further investigation.

* Abstract of paper submitted at Research Forum, University of Cape Town, 1 October 1958.

HYPERCALCIURIA WITH SPECIAL REFERENCE TO SARCOIDOSIS AND THE ACTION OF VITAMIN D*

W. P. U. JACKSON, M.A., M.D., M.R.C.P., *Department of Medicine, University of Cape Town*

Some abnormalities of calcium metabolism were considered, with particular reference to the hypercalciuria of sarcoidosis. The results of several metabolic studies were discussed. It appears that the 3 disturbances of calcium metabolism in sarcoid—hypercalcaemia, hypercalciuria, and sarcoid intestinal absorption—are to some extent independent of each other.

In 2 cases of idiopathic hypercalciuria the high urine calcium was also associated with a high intestinal absorption, very similar to the state of affairs in sarcoidosis when hypercalcaemia is not present. In both conditions a negative calcium balance is produced by using a low calcium diet.

The effects of vitamin D on calcium metabolism were briefly considered, and these appear to be mainly the same 3 as were found to occur in sarcoidosis. It is suggested that in these and in some other conditions (e.g. hyperparathyroidism, acromegaly) the hypercalciuria comes first and produces a stimulus to the intestine to increase its absorption of calcium. This may represent a new principle of body metabolism with eference to calcium.

The disturbances of calcium metabolism in sarcoidosis can logically be explained as sensitivity to vitamin-D effect; one difficulty being that not all patients appear to be sensitive. Luckily, all cases of this condition appear to respond to cortisone or its analogues, whether vitamin-D-sensitive or not.

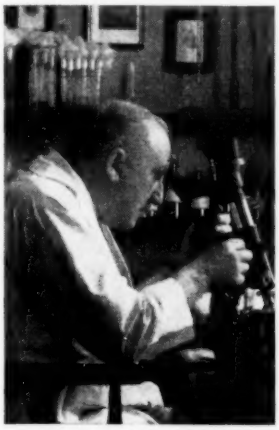
* Abstract of paper delivered at Research Forum, University of Cape Town, on 5 November 1958.

ASSOCIATION MEDALS

The following citations were read at the Adjourned Annual General Meeting of the Medical Association of South Africa held at Pretoria on 1 October 1958 when the Association's Silver Medal for Distinguished Service was presented to Prof. A. Pijper and the Association's Bronze Medals for meritorious Service to Drs. Schaffer, Schneider and Vercueil.

PROF. ADRIANUS PIJPER

Professor Pijper was born in Holland and studied medicine at the University of Leiden, in London, and at the School for Tropical Diseases at Hamburg. He came to South Africa in 1913 and after a few years in general practice on the High Veld came to Pretoria in 1920, where he established a private laboratory and was appointed Pathologist to the local hospitals and the Municipal Health Department. When in 1945 the Pretoria University started its medical faculty, he became its first Professor of Pathology and Director of its Institute for Pathology.



Prof. A. Pijper

Prof. Pijper had done extensive research work to improve diagnostic methods for typhoid fever, including the tracing of carriers. Through his work many outbreaks have been traced to their origin. His work in the field of detection of likely carriers has helped very much in the prevention of typhoid fever throughout South Africa. In a different sphere, he invented the diffraction method for the diagnostic measurement of red blood cells, for which an apparatus was built and marketed by Zeill. Another piece of research was the discovery that the new disease, tick-bite fever, was caused by a Rickettsia, which is named after him, and at the same time he showed the presence in South Africa of two other Rickettsioses, louse typhus and rat-flea typhus. He found ways and means to determine the blood groups of South African races, including Bushmen, Bantu and Hottentots. In mycology he established the unsuspected presence in South Africa of several infectious diseases caused by fungi. During the last 30 years recently with help from the Council for Scientific and Industrial Research, he has done much research into the problem of how bacteria swim. For this purpose he uses the sun as his microscope lamp and has made a number of cine films of moving bacteria through a microscope.

Prof. Pijper was awarded the Hamilton-Maynard Memorial Medal of the *South African Medical Journal*, the Scott Medal of the South African Biological Society, and the South African Medal of the South African Association for the Advancement of Science. He is an M.D. of Leiden University, a Fellow of the Royal Society of South Africa, an Honorary Fellow of the Royal Microscopical Society of London, and has an honorary D.Sc. degree of the Universities of Pretoria and Cape Town.

As a member of the Medical Association of South Africa, he has served on the Branch Council of the Northern Transvaal Branch and was a member of the Federal Council from 1931 to 1943. He is at present an Emeritus Member of the Association. He also served as an elected member of the South African Medical and Dental Council from 1942 to 1948.

Perhaps the most outstanding characteristic of this brilliant scientist is his almost fierce fanatical insistence on the highest integrity in scientific research. These impositions he placed upon himself and he will not tolerate the deviation from it by anyone else. Such standards must have had their influence on scientific work throughout the whole of South Africa, and for those who work with him it is a constant reminder of this man's belief in—almost worship of—the 'Truth'.

This is the man whom the Medical Association of South Africa wishes to honour by the award of its Silver Medal for Distinguished Service to medical science and humanity.

DR. RUDOLPH SCHAFFER

Dr. Schaffer was educated at Marist Brothers College, Uitenhage, Rhodes University College, the University of Cape Town, and Trinity College, Dublin. He graduated M.B., B.Ch. in 1924 and proceeded M.D. in 1929. Since that time he has done postgraduate work in Germany, London, Edinburgh, Dublin and the United States of America. He has been in general practice in Queenstown, C.P., since 1926, except during his absence on military service from 1940 to 1945. During the latter period he served at Premier Mines Military Hospital, Oribi Military Hospital, Ladysmith Military Hospital and at 107 S.A. General Hospital as a Specialist Physician. He was part-time Medical Superintendent of the Frontier Hospital, Queenstown for 8 years.

Dr. R. Schaffer

Dr. Schaffer joined the British Medical Association in Dublin in March 1924, and transferred his membership to South Africa in the same year. He joined the Medical Association of South Africa on its formation and has been a member since that date. He has been Chairman of the Queenstown Division 11 times and a member of the Queenstown Executive Committee since 1928. He has been President of the Border Branch on two occasions and a member of the Branch Council for many years. Dr. Schaffer was elected to Federal Council in 1947, since when he has been a member continuously and a member of the Executive Committee for the past 6 years. He has not failed to attend a South African Medical Congress since 1934.

Dr. Schaffer has spared neither time nor labour in the service of his colleagues, and his valuable counsel has long been indispensable in Border medical circles. In consideration of his long and distinguished service to the profession and to the Medical Association of South Africa, he is awarded the Bronze Medal for Meritorious Service.

DR. TOBIAS (TEDDY) SCHNEIDER

Dr. Schneider was born in London, England, and educated at Jeppe High School and the University of the Witwatersrand, where he qualified M.B., B.Ch. in 1927. Following the holding of various house appointments at the Johannesburg General Hospital and after a period of duty as Senior Resident Medical Officer at Boksburg-Benoni Hospital, he went into general practice for a period of 15 years. During this time he held various part-time positions at the University of the Witwatersrand as a demonstrator in anatomy and lecturer in the department of pharmacology. He was also on the honorary staff of the Johannesburg General Hospital, first as clinical assistant and then as medical registrar. He proceeded overseas, where he was a clinical assistant at St. Bartholomew's Hospital in London and at the Brompton Hospital for Diseases of the Chest. In 1940 he was made a Member of the Royal College of Physicians of Edinburgh.

During World War II he held the rank of Captain in the 25th Field Ambulance, S.A. Medical Corps. He was also Chairman of the General Practitioners' War Fund and a member of the Discharge Soldiers and Demobilization Committee in the Southern Transvaal.

Dr. T. Schneider

Since the war he has practised as a specialist physician in Johannesburg. He is assistant physician and physician in charge of the diabetic clinic of the Johannesburg Hospital and the University of the Witwatersrand. He is a past President of the Medical Graduates Association of the University of the Witwatersrand, he was Honorary Secretary of the Southern Transvaal Branch of the Medical Association of South Africa for a period of 5 years, and he was President of that Branch in 1952. He was a member of the Organizing Committee and Press Liaison Officer at the Medical Congress in Johannesburg in 1952. He has been Press Liaison Officer of the Southern Transvaal Branch for several years and is Chairman of the relative Committee as well as a member of the Ethical Committee.

Since 1946 Dr. Schneider has been a member of the Federal Council, and is at present Chairman of the Federal Ethical Committee. He was a member of the Steering Committee of the College of Physicians, Surgeons and Gynaecologists of South Africa, and is now on the Executive Committee of its Division of Medicine. For the past 3 years Dr. Schneider has served on the Augmented Executive Committee for the Transvaal. From 1953 to September 1957 he was Honorary Secretary of the Association of Physicians of South Africa, and is now its Vice-chairman. For the past 3 years he has been the Convener of the Liaison Committee of Federal Council with the Dental Association of South Africa.

For the services he has given to the cause of medicine in South Africa, and to the Medical Association of South Africa and its Southern Transvaal Branch, the Association has pleasure in awarding Dr. Schneider its Bronze Medal for Meritorious Service.

DR. LEON OLIVIER VERCUEIL

Dr. Vercueil was born in Middelburg, Transvaal, and having taken the first 2 years of his medical course at Cape Town he graduated M.B., B.Ch., B.A.O., at Trinity College, Dublin, in 1922. He received the D.P.H. and M.D. in 1924. He also holds the degrees of B.Sc. (Pretoria) and M.A. (Dublin). He has done postgraduate work in Dublin, London, Oxford, Liverpool, Vienna and Budapest. Since 1924 he has been practising on the West Rand.

Dr. L. O. Vercueil

Dr. Vercueil had a distinguished military career during World War II. He was Lieutenant Colonel in command of the 18th Field Ambulance throughout the North African Campaign.

Dr. Vercueil has been Chairman of the West Rand Division of the Southern Transvaal Branch for 9 years, a member of the Branch Council since 1939, and President of the Branch in 1954. He has been a member of Federal Council for the past 15 years and a member of its Central Committee for Contract Practice since its inception; he has been Chairman of this Committee for the past 4 years and Chairman of the Workmen's Compensation Committee of Federal Council, of the Mines Benefit Societies Medical Officers' Group and of the Railway Medical Officers' Group. During the dispute over the Ordinance to provide free hospitalization in the Transvaal, Dr. Vercueil spent a great deal of his time visiting and organizing country doctors, and as a result he was a tower of strength to the Association. Every member of the Association is indebted to Dr. Vercueil for his success as a negotiator on behalf of the Association with the Workmen's Compensation Commissioner, the Railway Sick Fund and the representatives of the Medical Aid Societies.

The Association greatly values the service of Dr. Vercueil and seeks to honour him by the award of its Bronze Medal for Meritorious Service.

MEDICAL COUNCIL ELECTION

Below are published particulars concerning a candidate for election to the South African Medical and Dental Council.

DE VILLIERS, BECK, B.A., LL.B. (S.A.), M.B., B.CH. (RAND) Dr. Beck de Villiers is in 1910 op Ficksburg gebore waar hy in 1926 matrikuleer het. Daarna het hy na die destydse Grey-Universiteitskollege (G.U.K.) gegaan om in 1929 die graad B.A. en in 1932 die graad LL.B. te verwerf. In die jare 1930-1933 was hy ook prokureursklerk en later regtersklerk. Vanaf 1933 was hy eers op Smithfield en daarna tot 1941 op Vereeniging prokureur. Begin 1941 het hy sy praktyk verkoop om medisyne te studeer. Die grade M.B., B.Ch. het hy in 1946 aan die Universiteit van die Witwatersrand behaal.

Vanaf sy skooljare het hy aktief aan kultuur- en verenigingslewe, asook aan sport deelgeneem—ook as organiseerder. Aan die G.U.K. was hy 4 jaar lank lid van die studenteraad en het hy o.a. deelgeneem aan die stigting van 'n junior wetsvereniging in Bloemfontein. Op Smithfield was hy lid van die dorpsraad. In 1945 was hy aan die mediese skool voorsitter van die mediese studenteraad.

In 1948 het hy hom as algemene praktisyn op Bloemfontein gevestig. Kort daarna het hy ere-sekretaris van die Sentrale Afdeling (Tak O.V.S. en B.) van die Mediese Vereniging van S.A. geword en in 1954 is hy as Voorsitter gekies. Vanaf 1948 is hy ook ononderbroke lid van die Takraad, vir die 6 jaar vanaf 1949 as ere-sekretaris en vanjaar as President. In 1954 was hy lid van Federale Raad. Tans is hy ook voorsitter van die plaaslike groep van algemene praktisyns.

NUTRITION AND FISHERIES RESEARCH

The General Conference of Unesco is holding its 10th session in Paris from 4 November to 5 December 1958. The Conference is attended by delegates from Unesco's 80 member States, and will discuss the programme for 1959-60, involving a spending budget for the two years amounting to over 25 million dollars. One of the programme activities is marine research particularly, on this occasion, in the Indian Ocean. A recorded 30 million tons of fish and plants are obtained every year from all the oceans, but it is believed that this harvest, which is one of the utmost world importance in human nutrition, can be greatly increased by study of the fundamental physical phenomena that govern underwater life, and the application of improved methods of fishing. Marine science—biology, physics and chemistry—is opening up new fields of marine food supply while at the same time ensuring

the preservation of their source. The necessity of extending this research is emphasized also by the agriculturists, who are questioning the ability of land husbandry to meet human food demands of the future.

The marine research programme of Unesco was started in 1955, when an International Advisory Committee on Marine Sciences was formed, with a nucleus of representatives from Australia, Cuba, Denmark, France, Japan, India, the UK, the USA and the USSR, and the addition of experts in marine science. This Committee meets yearly and the first meeting, held at Lima, Peru, in 1956, dealt with the S.E. Pacific Ocean area. As a result of this Committee's recommendations a tour was made in S.E. Asia by Dr. A. F. Bruun, Danish oceanologist, who found a great shortage of experts in marine science and trained laboratory staff in the S.E. Pacific countries. To meet this need Unesco has granted study fellowships every year since 1956, and the Committee has recommended a long-term regional training programme and the appointment of a regional adviser in marine science to make tours during 1960 as a consultant to national authorities in the setting up of regional programmes and the equipping of laboratories.

Unesco is also cooperating with the International Society of Physical Oceanography and the existing institutes in various countries, and has made grants to Norwegian and Danish organizations.

The challenge of the Indian Ocean belongs to the future. In a concerted effort 11 nations, under the chairmanship of Dr. R. R. Revelle, American oceanologist, will spend some years in preparatory work. Part of their plan is the building of a special research ship (according to present ideas, of 1,300 tons and about 250 feet long) with laboratory space and accommodation for scientists and students and the chartering of other vessels for ocean research on an international basis. The attention of member governments is to be drawn to the importance of marine research, and Unesco is to be asked to help by providing contracts for individual research workers and laboratories, granting training fellowships and travel grants, and assigning experts to give technical assistance to countries requiring such aid.

Bearing on this subject of the 'harvest of the sea' the Commission for Technical Cooperation in Africa South of the Sahara is calling an international meeting in Cape Town in 1959 to study fisheries problems of the Eastern side of the continent of Africa. A similar international meeting held in 1957 at Luanda, Angola, dealt with the same problems on the western coast of Africa south of the Sahara.

THE MEDICAL PROFESSION AND THE CUBAN DISTURBANCES

The Secretary General of the World Medical Association (Dr. Louis H. Bauer) has reported a recent visit to Cuba where, without involving members of the medical profession, he has made enquiries into the disturbed conditions in Cuba and their effects on the medical profession and medical services.

Dr. Bauer reports that since October 1957, when the World Medical Association intervened in the interest of humanitarian medical services for all people in Cuba, the situation has improved somewhat. The Cuban Medical Association is now permitted to hold meetings and to publish its journal. However, the medical profession continues to need the support of the World Medical Association in its efforts to provide medical services to anyone needing aid on the basis of caring first for those most in need of these services regardless of race, colour, creed or party politics. This is the duty and the right of every doctor under the Declaration of Geneva and the Red Cross Convention of 1949.

Dr. Bauer reports that the revolution is active, with actual fighting in all parts of Cuba except Havana and Matanzas. In the eastern provinces doctors are forbidden to give medical aid to revolutionists. If they do so, they are warned to leave within 2 hours or be killed. As a result, hundreds of doctors with their families have been forced to seek refuge in Havana, Mexico and the United States. Education is at a standstill. Even in Havana the university and public schools remain closed and the faculty members are unpaid.

The Secretary General appeals to the other 54 member associations of the World Medical Association to implement the provisions of the resolution for the support of the Cuban Medical Association adopted at the XIIth General Assembly in August 1958. He stresses the importance of basing this support upon the humanitarian precepts of medical service, which must be completely divorced from the political aspects of the situation. He notes that it is the duty of the members of the World Medical Association to insist that every doctor everywhere in the world must be protected in carrying out his humanitarian duties and that his right in so doing must be recognized.

INTERNATIONAL FEDERATION OF SURGICAL COLLEGES

The International Federation of Surgical Colleges was formally inaugurated on 4 July 1958 at Stockholm. This federation is representative of surgical colleges, academies and institutions throughout the world and is governed by a Council consisting of a President, 2 Vice-Presidents and a representative of each member body which is not represented by the President or Vice-Presidents. The Council will meet annually. The object of the Council is the improvement and maintenance of the standards of surgery throughout the world by methods such as the establishment and maintenance of close relationships among approved surgical colleges and institutions; encouragement of the maintenance of high standards of education, training and research in surgery and its allied sciences; the unification of surgery in all its specialities; the support of clinical and scientific congresses; contribution to peace in the world through friendship and promotion of understanding among surgeons of all nations; and the provision of surgery of high standard to all countries of the world requesting aid.

The president of the Federation is Prof. Sir Harry Platt, Bt., M.D., M.S., F.R.C.S. (UK); the Vice-Presidents are Dr. I. S. Ravdin, M.D. (USA) and Prof. E. Dahl-Iversen, M.D. (Denmark). The founder-member institutions are the following:

Académie de Chirurgie, American College of Surgeons, College of Physicians, Surgeons and Gynaecologists of South Africa, Collegium Regium Chirurgorum Universitatum Daniae (Royal College of University Surgeons of Denmark), Danish Surgical Association, Nederlandse Vereniging voor Heelkunde (Dutch Association of Surgeons), Norsk Kirurgisk Forening (Norwegian Surgical Association), Royal Australasian College of Surgeons, Royal College of Physicians and Surgeons of Canada, Royal College of Surgeons of Edinburgh, Royal College of Surgeons of England, Royal College of Surgeons of Ireland, Royal Faculty of Physicians and Surgeons of Glasgow, Societa Italiana di Chirurgia (Italian Surgical Society), Société Belge de Chirurgie (Belgian Surgical Society) and the Svensk Kirurgisk Förening (Swedish Surgical Society).

It is expected that other member-institutions will be added at the next meeting of the Council.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. T. Schneider, M.R.C.P. (Edin.), of Johannesburg, will have the degree of Doctor of Medicine conferred on him by the University of the Witwatersrand for his thesis on 'Observations on Diabetes Mellitus at the Diabetic Clinic of the Johannesburg General Hospital'. The degree will be conferred at the graduation ceremony on 15 December 1958.

Dr. H. van der Meulen, Dermatologist, 512 Medical Centre, Heerengracht, Cape Town, is now practising also as a Venereologist. Telephone 3-0403.

Dr. H. van der Meulen, Dermatoloog, Mediese Sentrum 512, Heerengracht, Kaapstad, praktiseer nou ook as Venereoloog. Telefoon 3-0403.

* * *

The American College of Chest Physicians (Cape Town Group) will meet at Medical Centre, Heerengracht, Cape Town, in the rooms of Dr. K. Brauer, on 1 December 1958 at 8.15 p.m. Dr. Maurice Nellen, who has recently returned from a visit overseas, will give an account of his impressions of the Third World Congress of Cardiology held in Brussels, which he attended. All practitioners will be welcome.

* * *

Paediatric Prize Essay. The South African Paediatric Association has awarded the Paediatric Prize for 1958 to Mr. A. B. Ockerse, a 5th-year medical student at the Pretoria University. The title of the essay is 'Pneumonia in the first two years of life'. A Bronze Medal has also been awarded to the same candidate.

The subject of the essay in this competition for 1959 is 'The aetiology of gastro-enteritis in infants'. The prize is an award of £10 and a Bronze Medal if the essay is of sufficient merit.

* * *

Medico-surgical Cinema: Prize awarded by 'La Presse Medicale'. The Annual Prize for Medico-Surgical Cinema, comprising 100,000 francs cash (with possibility of being divided) and various other awards, will be awarded during the last session of the Course of *Actualités Médico-Chirurgicales* at the *Nouvelle Faculté de Médecine de Paris* on 17 March 1959. The Jury will consider the didactic value of the film as well as its properly cinegraphic quality. Only the 16 mm. size film will be admitted. Entries and films are to be sent to the *Secretariat du Journal 'La Presse Medicale'*, 120 Boulevard Saint Germain, Paris VI°, before 15 February 1959. Awards will be remitted to the authors of the best films. Contrary to former years, films subsidized or produced by a laboratory or firm will not be excluded from the competition.

* * *

Dermatology and Venereology Group (Cape Town Sub-group). On Tuesday 25 November at 8.15 p.m. in E Theatre Groote Schuur Hospital, Cape Town, Dr. R. Kooij, M.D. (Amsterdam), recently appointed to the Department of Dermatology, Groote Schuur Hospital and University of Cape Town, will give a talk on 'The Kveim Reaction: its bearing on the Aetiology of Sarcoidosis', being an amplification of an address recently given before the Royal Society of Medicine, London. Medical practitioners and students are cordially invited.

* * *

The United Nations announce the forthcoming publication of the official English edition of the Proceedings of the Second International Conference on the Peaceful Uses of Atomic Energy, which was held at Geneva on 1-13 September 1958. This edition will consist of 34 volumes—page size 8½ by 11 inches, each volume about 500 pages, bound in Roxite vellum—which will be issued to subscribers as they become available between December 1958 and (probably) July 1959. The retail price will be about £5 5s. 0d. per volume (£182 the full set), but for orders placed by 30 November 1958 a prepublication price of £155 is offered (postage free), which may be paid as an immediate deposit of 10% and per subsequent monthly invoices. The order may be placed through local booksellers or through United Nations, New York (cable address Unations, New York). The publication includes two volumes on Isotopes in Biochemistry and Physiology, one volume on Isotopes in Medicine, two volumes on Biological Effects of Radiation and one volume on Radiological Protection.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

BELLERGAL(R) RETARD

Sandoz Ltd. announce the introduction of Bellergal Retard and supply the following information:

Bellergal retard tablets are a new long-acting form of the proven Sandos Bellergal. They contain twice the dose of active ingredients present in ordinary Bellergal. Thus, one-half the number of Bellergal retard tablets suffices to elicit the same therapeutic effect. Each tablet contains: total l-alkaloids of belladonna leaf (Bellafolin(R)) 0·2 mg., ergotamine tartrate B.P. 0·6 mg., and phenobarbitone B.P. 40·0 mg.

Bellergal retard tablets consist of three fractions—yellow, red and green—two of which have a delayed action, and become effective after 4 and 8 hours respectively. The full action is spread out over a period of 10-12 hours. The multiple release of active ingredients ensures even sedation throughout the day and night, and absence of temporary recrudescence of symptoms caused by a 'low' in drug action.

Mode of action. Bellergal retard blocks the passage of excessive stimuli from psyche to soma in psychosomatic (stress and functional) disorders.

Indications. Somatic manifestations of functional and stress disorders, in particular:

Menopausal syndrome, pre-menstrual tension, morning sickness, functional gynaecological conditions.

Effort syndrome (cardiac neurosis and neurocirculatory asthenia).

Gastro-intestinal hypersecretion and hypermotility (duodenal ulcer).

Psychosomatic skin disorders (neurodermatitis, pruritus, etc.).

Prevention of migraine.

Dosage. 1 Bellergal retard tablet mornings and evenings. Dosage may be increased to 3 tablets daily. but this should not be exceeded.

Presentation. Bottles of 20, 100 and 250 tablets.

Distributors. Alex. Lipworth Ltd., P.O. Box 4461, Johannesburg, P.O. Box 4838, Cape Town, P.O. Box 1988, Durban and P.O. Box 3188, Port Elizabeth.

POLARAMINE

Schering Corporation, USA, supply the following information:

The *South African Medical Journal* in a recent editorial[1] has drawn attention to the very real need for a long-acting antihistamine which produces few side-effects. By coincidence, Schering Corporation USA announces a new anti-histamine, Polaramine, which is the dextro-rotatory isomer of chlorpheniramine, and is reported to be relatively free of side-effects, particularly the troublesome sedative action.

Polaramine is put up as a long-acting drug, in the well-known Repetab form, i.e. 3 mg. in the outer layer for prompt effect, and 3 mg. in the inner core for release 4-6 hours after ingestion. (Note, Polaramine is also available as a simple-dose tablet of 2 mg.)

Indications are prevention and treatment of all allergic conditions responsive to antihistamines, e.g., hay fever, vasomotor rhinitis, angioedema, urticaria, drug and serum reactions, food allergies, atopic and contact dermatitis, allergic eczema, pruritus ani and vulvae, pruritus of non-specific origin, and insect bites, as well as in selected cases of migraine headache and asthma.

Dosage. Polaramine 2 mg. tablets, one tablet 3-4 times daily. Polaramine Repetabs (usual dosage), one 6 mg. tablet in the morning and repeated in the evening before retiring. More refractory cases may require a third Repetab.

Packings. Polaramine Repetabs 6 mg., bottles of 20 and 100. Polaramine tablets 2 mg., bottles of 30. 100 and 1,000.

Further information may be obtained from Scherag (Pty.) Ltd., P.O. Box 7539, Johannesburg.

1. Editorial (1958): S. Afr. Med. J., 32, 844 (23 August).

SANDOSTEN(R) NASAL SPRAY

An error occurred in the second paragraph of the article under this heading on p. 1010 of the *Journal* of 11 October 1958. This paragraph should have begun as follows: Sandosten Nasal Spray contains thenalidine digluconate 0·5%, calcium lactobionate 17·5%, and ephedrine HCl 0·9%.

REVIEWS OF BOOKS : BOEKRESENSIES

DETERMINATIVE BACTERIOLOGY

Bergey's Manual of Determinative Bacteriology. 7th Edition. by Robert S. Breed, E. G. D. Murray, Nathan R. Smith assisted by 94 contributors. Pp. xviii + 1094. 120s. London: Baillière, Tindall and Cox Ltd. 1957.

The new edition of Bergey's manual has been divided into two books. The first volume, under the title 'Bergey's Manual of Determinative Bacteriology', contains an outlined classification of all known bacteria, parasitic and non-parasitic, and a description of the species and sub-species with appropriate keys. The second volume is called 'Index Bergiana', and contains known details of all inadequately described species, such as were described under the title 'Appendices' in previous editions. The result is that the manual is smaller in size and more convenient to handle for routine work, the second volume being more valuable to the research worker.

The number of contributors to the Manual has been increased from 60 in the last edition to 100, and also represent opinions from 14 different countries, so that it is more international in character.

The basis of classification of the schizomycetes has been altered in many respects. For example, the order eubacteriales has been divided into 2 groups, depending on the arrangement of their flagella: the pseudomonadales with monotrichate flagella, and the eubacteriales with peritrichate flagella. This method of subdivision may not meet with universal approval. The actual descriptions of the various organisms, parasitic and non-parasitic, has been increased.

The classification of viruses as given in the 6th edition has been omitted, the editors considering that a formal classification is inappropriate in view of the present state of knowledge about these strains.

At the end is a key for the identification of organisms from a practical point of view (not a classification). At first glance this appears to consist of non-parasitic or auto-trophic strains, but it does prove of use in the identification of the more rarely isolated strains found in a medical routine laboratory.

P.D.

OBSTETRICS AND GYNAECOLOGY

Recent Advances in Obstetrics and Gynaecology. 9th Edition By Aleck W. Bourne, M.A., M.B., B.Ch. (Camb.), F.R.C.S. (Eng.), F.R.C.O.G. and Leslie H. Williams, M.D., M.S. (Lond.), F.R.C.S. (Eng.), F.R.C.O.G. Pp. vii + 348. 78 Figures. 38s. net. London: J. & A. Churchill Ltd. 1958.

In keeping with previous usage in the publication of a new edition of this popular volume, some chapters of the 8th edition, including, regrettably, that very good one on stress incontinence, have made way for new material in this, the 9th. There is an excellent chapter on uterine function in pregnancy and labour, which is recommended to undergraduate and postgraduate students alike for its clear exposition and the comprehensive survey of historical and recent research work in this field. The anaemias of pregnancy are dealt with in detail in the other of the 2 new long chapters, a source of valuable information on this important aspect of antenatal care. Two of the 4 new short chapters are on the uncommon, but very worrying, complications of anuria and blood coagulation defects respectively. They make excellent reading in their clear exposition of pathogenesis and management. The former chapter is well illustrated by a graphic representation of the biochemical changes in anuria and recovery from it. The chapter on 'Changing Trends in Obstetrics' leaves one with a pleasant after-glow—obstetrics has certainly become simplified. Everybody is doing Caesarean sections for everything, so why worry about considering alternative measures or indulging in self-criticism after performing the operation for indications that are not so cut-and-dried? The operation is mentioned no fewer than 14 times in 2 pages of print under the heading 'Second Stage'. Having picked out this chapter for criticism, one cannot omit regretting that the wellknown technique of pudendal block is again described in detail, whilst no mention whatever is made of the value of caudal and extra-dural anaesthesia in obstetrics.

The possession of the full range of 9 editions of 'Recent Advances in Obstetrics and Gynaecology' is a must for every department engaged in this speciality, if not for every gynaecologist. Over the more than 30 years that this little volume has proudly taken its place on the shelves of medical libraries throughout the English-speaking world, very few subjects in its scope have not been assimilated in the various editions and its encyclopaedic value is inestimable, not only for the latest information but also for the historic value of the first editions. One has to read again the mortality figures for Caesarean section given in the first edition to appreciate in its true perspective the warning given by the authors in 1958: 'But if ever the haemolytic streptococcus should reappear in its old power and majesty, there may have to be an abrupt change back to more cautious methods and a full reappraisal of the position of Caesarean section.'

J.P R.

VERTIGO AND DIZZINESS

Vertigo and Dizziness. By Bernard J. Alpers, M.D., Sc.D. (Med.). Pp. vii + 120. 4 Figures. $5.00. New York and London: Grune & Stratton, Inc. 1958.

This small book is a monograph on a symptom, although one due to a disturbance of a special function, and as such it suffers from the defects inherent in attempting to be comprehensive without being prolix and even at times a little confusing. The author deals with his topic in a systematic way, starting with the anatomy of the vestibular system, its physiology, and then the mechanisms, symptoms and causes etc. of the symptom and he quotes quite a great deal from the literature in the course of his discussion. There are, however, times when one is left a little uncertain about his own views and which of the quotations he is in agreement with and which not. The last few pages deal with treatment which includes the diets recommended in Meniere's syndrome and one is left with the impression of optimism about their efficacy which practical experience hardly justifies, especially in a condition the natural history of which may be very variable with so many remissions and exacerbations. These diets have not the slightest effect in the severe and progressive case. The bibliography is a complete one and has the advantage of specially marking the best references.

S.B.

NEUROSURGERY

Clinical Neurosurgery. Proceedings of the Congress of Neurological Surgeons, Chicago, Illinois, 1956. Pp. x + 142. Figures. 56s. net. London: Baillière, Tindall & Cox Ltd. 1957.

'Diabetes Insipidus does not depend on the presence of the anterior lobe of the pituitary.' 'Probably the only criterion for the completeness of hypophysectomy is dependence on cortisone.' These are some of the findings of Bronson S. Ray and Olof Pearson in the chapters reviewing 110 cases of hypophysectomy for carcinoma of the breast. Surgeons, endocrinologists and neurosurgeons will find these chapters of the greatest interest.

The late Gilbert Horrax's article on modern radiotherapy of pituitary tumours may prove to be revolutionary. Further results with the use of the 2 million volt apparatus are awaited as in his present series only 12·1% of patients had to be operated on. Wilder Penfield, to whom this volume is dedicated, gives a fine exposition on the functions of the temporal lobe and, for students of the history of medicine, a description of his archeological investigations into the life and time of Hippocrates.

Altogether a valuable book and highly recommended.

A.G.

ARTIFICIAL LIMBS

Artificial Limbs. By Leon Gillis, M.B.E. Pp. xiv + 449. Illustrations. £15 net. London: Pitman Medical Publishing Co. Ltd. 1957.

Mr. Gillis has again performed a herculean task in writing this book. The effort involved was obviously prodigious and prolonged; it is all the more creditable since it was done while engaged in active orthopaedic practice. This book is the companion volume to his well-known text-book: 'Amputations'. He acknowledges the help of many individuals; this is typical of his modesty.

But, without his own highly specialized knowledge of this subject, this book could not have been written. The 3 sections cover every possible facet in great detail. There are very few criticisms that one can level at this tremendous piece of work with its remarkable number of references. I found the author's habit of repetition when stressing important points a little irritating. The style of writing is rather heavy and made reading difficult. These are, however, points of minor importance.

This book is a valuable addition to the great orthopaedic text-books of reference.

M.S.

PHYSICS FOR THE ANAESTHETIST

Physics for the Anaesthetist. Including a Section on Explosions. 2nd Edition. By Sir Robert Macintosh, D.M., F.R.C.S.E., F.F.A.R.C.S., M.D., William M. Mushin, M.A., M.B., B.S., F.F.A.R.C.S. and H. G. Epstein, M.A., Ph.D., F.F.A.R.C.S. Pp. xi + 443. 379 Figures. 60s. net. Oxford: Blackwell Scientific Publications. 1958.

The first edition of this book appeared in 1946 and in the field of books on anaesthetics, must surely be entitled to the term 'best seller'. So popular did this work become that a second edition was necessary the following year. The book has been out of print for the past 6 years and during this period, fortunate possessors of the first edition have found themselves constantly lending their copies to colleagues. Second hand copies have been unobtainable.

There is little doubt that this second edition will find a ready market, and this it clearly deserves. In common with all the books put out from the Nuffield Department of Anaesthetics in Oxford, it is a masterpiece of combined pictorial and literary presentation of its subject matter.

The main addition to this edition is a section on anaesthetic explosions. It is a fascinating section and probably contains more basic information on this subject, presented in a form easy for the average anaesthetist to understand, than is obtainable elsewhere. Another new chapter is that on pressure-reducing valves. This is a most welcome addition and here too, as is the case throughout the book, rather complex theory has been presented in simple words and figures, and even simpler diagrams.

While this book is designed principally for anaesthetists, there are parts which should be of benefit and interest to doctors in nearly all branches of medicine. Such things as the physics of syringes and needles, intravenous drips, the laws governing the flow of fluids and gases through tubes, which have so much bearing on the physiology of circulation and respiration, and a host of whys and wherefores of physics in everyday medicine are presented accurately as light reading, with illustrations which make for clear understanding.

This excellent book is in a class of its own both regarding its subject matter and its presentation.

A.B.B.

CORRESPONDENCE : BRIEWERUBRIEK

CONGENITAL TRACHEO-OESOPHAGEAL FISTULA

To the Editor: Mr. David Chapman, Senior Lecturer, Department of Surgery, Faculty of Medicine, University of Natal, was good enough to write me a letter drawing my attention to a very serious error in my article[1] on this subject, which was published in the *Journal* of 27 September 1958.

In the paragraph under the heading *Pathological Classification* Vogt's classification is described and the last passage of that paragraph is as follows: 'Of the published cases 90% fall in Vogt's group 3 (*a*). Of our 34 cases all but one, which fell into group 2, belonged to group 3 (*a*) . . .' In this passage 3 (*a*) should of course read 3 (*b*) in both places where it occurs. Reference to the diagram in the published article will explain this error.

Such is coincidence that this month we have had 4 cases of congenital tracheo-oesophageal fistula in Ward 9 of the Children's Hospital, Johannesburg, at the same time. Two of these had fistulae between the trachea and the upper pouch. These are the first two that we have had in over 40 cases personally seen.

Clarendon Centre
Park Lane, off Clarendon Circle
Parktown, Johannesburg
22 October 1958

David Adler

1. Adler, D. (1958): S. Afr. Med. J., 32, 958.

DIE ONDERRIG VAN KLINIESE ELEKTROKARDIOGRAFIE

Aan die Redakteur: Sal u my toelaat om 'n paar opmerkings te maak i.v.m. u hoofartikel in die *Tydskrif* van 18 Oktober oor bogemelde onderwerp.

U skep in hierdie artikel die indruk dat E.K.G.-dienste op die platteland van 'n lae gehalte is. U sê bv. ‚Die apparaat word in die gebruik gestel en die grafieke word gelees en geïnterpreteer op die grondslag van selfaangeleerde kennis wat dikwels gehaal word uit die talle klein en ondoeltreffende brosjures wat uitgegee word.'

Dat daar sulke mense bestaan wil ek nie betwis nie, maar hulle moet 'n klein minderheid uitmaak. In hierdie area (Suidwestelike Distrikte) is al die praktisyns wat toestelle het, mense met jarelange ondervinding in die praktyk, wat 'n intensiewe studie van die onderwerp gemaak het, en beslis nie op hol sal raak met 'n diagnose op sterkte van 'n elektrokardiogram nie.

Ek dink u kan aanneem dat meeste dokters wat toestelle koop, mense is wat hulle deeglik vóór die tyd voorberei het en nie soos u beweer, eers toestelle koop en dan uit brosjures leer hoe om te interpreteer nie.

Nou stel u voor dat studente beter onderrig moet word in hierdie onderwerp. So ver so goed. Maar as die toestand gaan ontstaan dat 'n dokter ná kwalifikasie die idee gaan hê dat hy nou in staat is om E.K.G.-werk in die praktyk te gaan doen sonder verdere studie, dan sou die toestand oneindig gevaarliker word as wat u wil voorgee dat dit nou is.

Ten slotte wil ek net sê dat u stelling ‚Almal besef bv. nie dat 'n miokardiale infarkt meer akkuraat gediagnoseer kan word op grond van die geskiedenis en fisiese bevindings . . . as wat dit met die kardiogram gemaak kan word nie', eenvoudig nie waar is nie.

Dat 'n pasiënt 'n infarksie kan hê met 'n normale E.K.G. is moontlik alhoewel dit nie alte dikwels in die praktyk voorkom nie. Maar ons het al almal gevalle teengekom waar die kliniese toestand (veral met 'n klein infarksie agter in die hart) baie twyfel laat ontstaan waar die E.K.G. absolute bewys lewer en alle twyfel verwyder.

B. M. Nel

Posbus 34, George, K.P.
5 November 1958

1. Van die Redaksie (1958): S. Afr. T. Geneesk., 32, 1016.

A MISSING VOLUME

To the Editor: I wish to encroach upon your valuable and always limited space in order to recover volume 7 of my set of British Encyclopaedia of Medical Practice (Sir Humphrey Rolleston) volumes.

Some years ago I was lucky enough to deal with a case of Otto's pelvis complicating delivery at the New Somerset Hospital. The only reference I could find to the condition was under the orthopaedic section of volume 7, page 305 of the B.E.M.P.

I unfortunately lent my bookplated volume 7 to a young obstetrical enthusiast, who has not returned the book. I am unaware who the culprit is.

May this letter, my bookplate and your kind assistance help me to recover the lost volume.

D. P. de Villiers

Robb's New Building
Belmont Road, Rondebosch, Cape
11 November 1958